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VOL. VII.

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BRAIN.

APRIL, 1884.

Original Articles.

HEMISECTION OF THE SPINAL CORD.

BY DAVID FERRIER, M.D., F.R.S.

THAT the paths of voluntary motor impulse in the spinal cord are mainly, if not exclusively, direct, and those of sensation crossed, is the hypothesis which accords best with the facts of disease in man. There may be differences among the lower animals as regards the totality of the decussation of the motor tracts in the medulla oblongata, and of the sensory tracts in the cord itself, but the general law first laid down by Brown-Séquard has been fully confirmed by all the more recent and carefully conducted physiological experiments. While the paths of voluntary motor impulse have been satisfactorily defined by a combination of experimental, embryonic and pathological research, those of sensation have not been determined with the same degree of accuracy.

The most careful experiments on the lower animals, such as those of Ludwig and Woroschiloff, always leave room for doubt whether the reactions which result from the application of sensory stimuli to parts below the experimental lesion are to be regarded as signs of sensation proper, or merely of reflex action more or less general. On questions involving subjectivity the most satisfactory evidence would be furnished by observations in man, but we rarely if ever meet with spinal lesions so exactly limited as to be made available for this purpose. Of the numerous cases of unilateral spinal lesion on record, there is scarcely one of which, but for the aid of experimental physiology, it could be said that the unilateral character of the lesion was at all certain. Most of them have not been investigated post-mortem, and of those that have been so, the investigation has been so imperfect, or the lesions have been so indefinite, that the unilateral character of the lesion has been assumed rather than proved.

I have only found one case in which the lesion was sufficiently definite and carefully investigated post-mortem; and even in this case the lesion was not strictly unilateral. The case in question has been described by W. Müller (Beiträge z. Pathologie und Physiologie des Rückenmarks, 1871). The patient was a woman who received a stab with a knife which traversed the spinal cord in a somewhat oblique direction below the third dorsal nerve, completely severing the left half of the cord, and also the right posterior column as far as the junction of the posterior root on that side. The division of the right posterior column therefore complicated a case otherwise definitely unilateral.

With the view of determining the effects of strictly unilateral division of the spinal cord in an animal as nearly human as possible I made the following experiment on a monkey.

The animal was chloroformed deeply, and, under stringent antiseptic surgical precautions, the cord was exposed in the region of the seventh and eighth dorsal segments.

Midway between the seventh and eighth nerves the left half of the cord was severed with a fine tenotomy knife. The incision was made, with the aid of a magnifying glass, from the posterior fissure outwards, without touching the right posterior median column.

The wound was carefully sewn up and dressed with antiseptic gauze and bandages.

The results described below were ascertained by daily observation and repetition of tests for a fortnight after the operation. During this time the general health of the animal was in the most satisfactory condition. On the fifteenth day the wound was examined, but it was found not to be united, though perfectly aseptic and free from discharge. It was again sewn up and dressed. Three days subsequently to this the animal, which had continued apparently in good health, died suddenly.

On examination after death, an extensive clot was found pressing on the anterior surface of the pons and medulla oblongata. The hæmorrhage appeared to have originated from some of the lateral branches of the basilar artery.

The wound in the dorsal region was more or less gaping, but though there was some discharge in the dressings, the bottom of the wound and the spinal canal were perfectly free from pus. The pia-mater was somewhat thickened and adherent around the region incised, but the cord, with the exception of the lesion described below, was entirely free from injury or inflammatory softening.

The results of the lesion were as follows:—

On the side of lesion (left).

Mobility.—The left leg was absolutely paralysed as to motion, and not the slightest movement was made with this limb either independently or in association with the right leg. The leg was dragged when the animal moved about its cage with the aid of the hands and the right leg.

The temperature of the left leg was, during the first two days, much higher than that of the right. No exact thermometric determination was however made. When this was attempted on the fourth day, the difference, judged by the hand, was barely, if at all, perceptible, and the clinical thermometer, not graduated below 90° F., did not register on either side.

The patellar tendon reaction was at first greatly impaired or entirely abolished. While it was distinct on the right, none could be elicited on the left. Before death, however, the left patellar reaction was much more distinct than the right.

Sensibility.—The slightest touch or ruffling the hair of the left foot, flank, or left side of the tail, invariably attracted the animal's attention and caused it to look, put its hand to the part, or move out of its position as if wishing not to be disturbed. A prick with a pin, a pinch, or most of all, a touch with a heated wire caused it to rub the part, or angrily try to seize and bite the offending cause.

Repeated comparison of the reaction to painful stimuli of the hands with that of the left leg, did not however indicate any such difference as could be construed into the existence of hyperæsthesia of the left leg. If it existed, it was not shown objectively.

On the opposite side (right).

Mobility.—The right leg was freely moved volitionally in all directions, and for all purposes, without any appearance of uncertainty or ataxy. The animal could grip with its right foot with considerable vigour, and did so as well as with its hands, when e.g. it wished to free itself when held prisoner.

This however was true only when vision was free, and no impediment was offered to the intended movement of the right leg. When the animal was held prisoner and its eyes blindfolded, it was utterly unable to extricate its right leg from any opposition made to its intended movement, though as soon as the eyes were uncovered, it used the foot to grip and endeavour to free itself. The same inability to free the leg without the aid of vision was seen when the animal was crossing a ledge through a small door leading into another compartment of its cage. Happening to catch its toes on the ledge, it could only extricate the foot on looking round to the cause of the obstruction.

The manifest difference between the use of the right leg, with and without vision, clearly indicated the abolition of the sense of muscular contraction, and inability to appreciate the position assumed by the leg, except with the aid of vision.

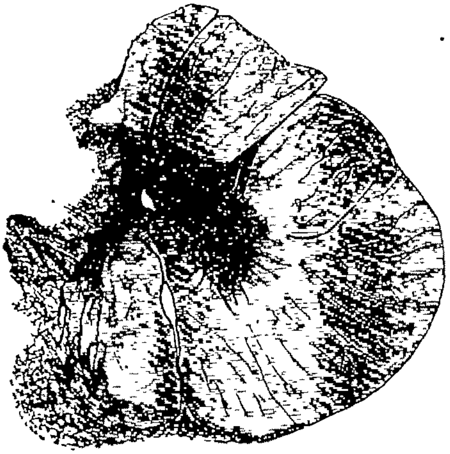
Sensibility.—In addition to the abolition of the so-called muscular sense indicated in the preceding paragraph, there was total absence of any sign of perception of contact, ruffling the hair, pricking, pinching the toes or the belly of the muscles, or the application of a degree of heat sufficient to excite the most lively demonstrations of sensation in the hands or left leg.

Not unfrequently, the momentary application of a heated wire to the toes or sole of the right foot was followed by a quick sharp movement of withdrawal. This was evidently purely reflex action, as the animal continued after this to allow the wire to remain in contact with the foot quite unconcernedly, though its transference to the hands or the left foot at once elicited lively demonstrations of pain.

There was thus total anæsthesia and analgesia of the right leg; and this extended to the right flank and right side of the tail.

The power of evacuating the contents of the bladder and rectum was not perceptibly affected.

We have thus in this case complete motor paralysis, with retention of sensibility on the side of lesion, and complete anæsthesia and analgesia with retention of mobility on the side opposite the lesion.



The lesion.—The cord, otherwise intact, was found to be divided on the left side between the seventh and eighth dorsal nerves. The edges of the incision were slightly everted and sharply cut. No softening existed. The cord was hardened in bichromate solution before the exact extent of the incision was further investigated. In order to determine how far inwards it extended, the portion of cord including the lesion was immersed in a strong solution of logwood. This penetrated the lips of the incision, and tinged all the divided surfaces.

The transverse incision was then completed, and the exact extent of the primary incision accurately determined.

In the preceding figure (p. 5), which is a drawing by Mr. Collings, from a micro-photograph (magnified 12 diam.) of the first slice from the cord above the incision, the exact extent of the lesion is clearly displayed. The whole of the right side is intact. The posterior fissure is distorted to the right, so that the line joining the anterior and posterior fissures, instead of being straight, forms an arc of a circle with the convexity to the left. The left anterior column immediately bounding the anterior fissure—the anterior median column—is intact as far outwards as a line drawn from the anterior surface of the cord parallel to the anterior fissure through the apex of the anterior horn. A portion also of the anterior root zone external to this line was left undivided, but was almost severed from the anterior median column.

A wedge-shaped portion—the base anteriorly—of the left posterior column immediately bounding the posterior fissure was also undivided. The whole of the columns of the left side of the cord, with these exceptions, were severed. The blackened irregular-looking remnant, seen on the left side, is due to the logwood staining and irregularities of the divided surface. The grey matter round the central canal as well as the anterior commissure, and some portions of the grey matter of the anterior and posterior horns, were uninjured.

The internal boundary of the divided surface of the left half of the cord would correspond with a line drawn parallel to the anterior fissure, through the apex of the anterior horn, and gradually approaching the posterior fissure.

Microscopical investigation of sections of the spinal cord above and below the lesion, for which I am indebted to the assistance of my colleague Dr. Beevor, showed ascending degeneration, characterised by atrophy of the axis cylinders without any increase in connective tissue, in the left column of Burdach and in the direct cerebellar tract. For a couple of segments above the lesion an irregular wedge-shaped area, the base posteriorly, was visible in the column of Burdach less stained by carmine than the rest, owing to the almost entire absence of axis cylinders. In the cervical region the column was however entirely normal. No degeneration was visible in the column of Goll immediately above the lesion, or in the cervical region, this portion of the posterior column having entirely escaped section or subsequent secondary lesion. There was extensive disappearance of axis cylinders, without sclerosis, in the left direct cerebellar tract above the lesion, and clearly visible also in the sections of the cervical region.

Below the lesion there was disappearance of axis cylinders in the postero-lateral column of the left side, corresponding to the area of the crossed pyramidal tract. No degeneration could be detected in the direct pyramidal tract, or in any of the other columns.

As in this case the right posterior column was absolutely intact, it is apparent that Schiff's doctrine that the posterior column is the path of tactile sensation on the same side, is erroneous. As the grey matter of the right side and also that surrounding the central canal on the left side was intact, it is also plain that these are not sufficient to convey the sense of pain on the right, for there was the most complete analgesia of the right leg. The experiment therefore is in absolute contradiction to the doctrines of Schiff, and entirely harmonises with those of Ludwig and Woroschiloff, which place the paths of voluntary motor impulse for the same side, and those of sensation for the opposite side in the lateral columns.

The motor path, the pyramidal tract, has been well defined; that of sensation, and its relations to the posterior horn and the tracts in its neighbourhood, require yet further elucidation.

If we compare the symptoms above described with those which are commonly said to result from unilateral lesion of the spinal cord in man, we find many points of agreement, but at the same time certain points of difference of an important nature and specially worthy of comment.

It is laid down by Brown-Séquard ('Lancet,' vol. ii. 1869, p. 869), who has been followed almost without question by most writers and text-books, that on the side of lesion there is paralysis of voluntary motion and of the blood-vessels, retention, in an exalted degree, of sensibility to touch, tickling, pain, heat and cold, but loss of the muscular sense; while on the opposite side there is conservation of voluntary motion and of the muscular sense, with total anæsthesia and analgesia otherwise.

As regards the total paralysis of voluntary motion and retention of sensibility on the same side, and retention of voluntary motion with anæsthesia and analgesia on the opposite side, the results of the above experiment are in complete harmony.

The fact of vaso-motor paralysis also on the side of lesion was confirmed by the evident increase of temperature in the left leg. This, however, was not persistent, in the same degree at least. Though the point was not investigated very minutely, there was (a few days after the operation) a very marked diminution of the difference in temperature between the two limbs which was at first so apparent. This would lend support to the views of Goltz and Vulpian, that the tone of the blood vessels is not entirely dependent on the vaso-motor centre of the medulla oblongata, but is capable of being maintained by the spinal centres themselves.

The existence of hyperæsthesia on the side of lesion was not evidenced by any noteworthy difference in the vivacity of response to sensory stimulation in the hand and foot respectively. But, on this point, the observations in man are so much superior to any inferences from the lower animals, and the existence of hyperæsthesia on the side of lesion has been so generally observed, that I should not regard the experimental facts above narrated as opposed to, even if they do not support, the existence of a condition of heightened sensibility.

As regards the muscular sense, however, the experiment is in diametric contradiction to the assertion that this sense is retained on the side opposite the lesion; and so far as I have been able to discover by examination of the published cases of hemispinal lesion, the assertion that the muscular sense is lost on the side of lesion, and retained on the opposite side, is not supported by any satisfactory evidence.

Most of what has been regarded as evidence of the muscular sense on the opposite side has no such meaning, and is of no value whatever in this relation. By the term muscular sense, is properly meant the knowledge we have of the degree of strain on our mobile parts, and of the position assumed by them actively, or given to them passively. It is usually tested by the power of discriminating different weights or resistances, or by the capacity of judging blindfold the position in which a limb is during active or passive movements. It has however been regarded, by Brown-Séquard and others, as sufficient evidence of the muscular sense in the non-paralysed limb, that the individual is able to direct his movements aright. Thus of one of his cases he says, "the power of directing movements, which depends on the muscular sense, was markedly altered in the paralysed lower limb, while it was perfect in the non-paralysed limb."[1]

The power of directing movements, however, has no necessary connection with the muscular sense, and may be perfectly well retained when there is not a vestige of the muscular sense. During the acquisition of perfect volitional control over our limbs, the sensations arising in connection with muscular action no doubt largely help in guiding and directing our movements, but the aid of vision is also relied on to a much greater extent than is usually supposed.

The power of directing movements, once acquired, is capable of being exercised without any sense of muscular contraction whatever, and also without the aid of vision. Whether the intended movement has been actually carried out, however, the individual paralysed as to muscular sense is not aware unless his eyes are free.

In cerebral hemianæsthesia, even without the aid of vision, the patient is able to intend and carry out a given movement fairly well if no obstruction is offered. There is a remarkable case on record which proves that also, in total anæsthesia of spinal origin, the power of directing and duly co-ordinating the movements of the limbs is perfectly retained in the total absence of all sense of muscular contraction, and even with the eyes shut. In this case, recorded by Schüppel,[2] there was, in consequence of syringo-myelitis, total anæsthesia, and total abolition of muscular sense in the limbs, and yet the man could move his limbs vigorously, and carry out any desired movement with his eyes shut, without exhibiting any greater degree of hesitation or caution than a perfectly normal individual would exhibit under similar conditions.

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[1] 'Lancet,' vol. ii., 1868, p. 594.

[2] 'Archiv d. Heilkunde,' vol. xv., 1874, p. 44.

It is evident from these and similar facts, that the sense of muscular contraction is entirely separable from the motor innervation of the muscle. It is a complex of centripetal impressions generated by movements, active or passive, in the skin, joints, tendons, fasciæ and muscles themselves, in virtue of the sensory nerves with which these parts are furnished. That the whole of these nerves enter by the posterior roots and ascend in the cord entirely distinct from the paths of motor impulse has been proved anatomically, as well as by experimental and pathological evidence.

But, inasmuch as in cases of partial lesion of the sensory tracts there may be anæsthesia without analgesia, and analgesia without anæsthesia, it is theoretically possible that notwithstanding cutaneous anæsthesia, the sensibility of the muscles and deeper parts may continue, and thus form the basis of a sense of muscular contraction. But such a condition has never occurred in my experience, and I have nowhere been able to find satisfactory evidence to that effect. There is good reason for believing that, in all cases where the muscular sense has been retained, notwithstanding the abolition of the sense of pain and very great diminution of tactile sensibility, the power of localisation of cutaneous sensory impressions has remained acute. That this is possible has been well shown in a carefully investigated case recorded by J. Mackenzie,[1] in which, in spite of analgesia and anæsthesia to various forms of stimuli, the patient was able to localise with accuracy the slightest touch on the anæsthetic limb.

In a case reported by Brown-Séquard (Case 3, 'Lancet,' sup. cit.), in which it is stated that muscular sense remained notwithstanding the existence of almost complete anæsthesia and analgesia, it is expressly stated that the power of localisation continued perfectly accurate.

In Mackenzie's patient, when, at one period in the history of the case, the anæsthesia was absolute and the power of localisation abolished, the muscular sense was also entirely annihilated.

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[1] A case of Hemiparaplegia Spinalis, 'Lancet,' June 9 and 16, 1883.

But, even if this power of localisation were inadequate to account for the existence of the so-called muscular sense in a limb otherwise anæsthetic, the retention of the muscular sense would, in view of the facts above related, indicate merely that the sensory tracts of the opposite side of the spinal cord had not been entirely interrupted, and would lend no countenance to the extraordinary doctrine enunciated by Brown-Séquard, that the paths of the muscular sense lie in the anterior roots and motor tracts of the spinal cord. This doctrine implies, and explicitly states, that the muscular sense is lost with the power of motion on the side of lesion. If by the muscular sense, we mean merely the power of directing movements, then, of course, this is lost on the paralysed side; but the loss is of motor power only, and not of the sense of movement. Naturally a patient cannot be made to estimate weights by volitional movements of a paralysed limb, but that he retains the muscular sense, so far as regards his knowledge of passive movements communicated to his limb, has been demonstrated beyond all doubt.

Few of those who have reported cases of unilateral disease of the spinal cord seem to have applied any proper tests on this point. But Köbner, in a case he has recorded,[1] found that in the paralysed limb, the perception of every movement communicated to it was perfectly retained, and the electro-muscular sensibility was in every respect normal. In Mackenzie's case also, where the limb was not entirely paralysed, the patient could appreciate weights quite correctly, so far as his feeble powers permitted; though he made mistakes when he attempted heavy weights, owing to the excessive sense of effort thus secondarily induced.

Brown-Séquard's dictum, therefore, that the muscular sense is lost on the side of lesion, is absolutely contradicted by facts such as these.

That the paths of muscular sense have any relation whatever to the anterior roots and motor tracts is opposed to every well-established fact of anatomy, physiology, and pathology of the spinal cord. Schüppel's case proves to demonstration that the paths of muscular sense, and sensibility in general, can be entirely interrupted, while those of voluntary motion remain intact. The motor tracts and anterior roots in this case were perfectly normal.

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[1] Deutsches Archiv f. Klin. Med., Bd. xix., 1877, p. 169.

That the muscular sense can be exercised, even to exact discrimination of differences in weight altogether independently of motor innervation, I have elsewhere shown.[1] In certain experiments made by Dr. Lauder Brunton and myself, it was found that the discrimination of weights when the muscles were made to contract artificially by the galvanic stimulus, was about as good as when the limb was moved volitionally.

As regards the mere knowledge of the position which a paralysed limb is made to assume by passive movements communicated to it, this, as Köbner's case shows, may be perfectly retained when the motor tracts of the cord are divided on one side. And it will doubtless be found, if the matter is investigated, that under such circumstances the patient will be able to discriminate differences in weight, by arrangements for exciting the muscles to action by electrical stimulation. But, indeed, so far as the knowledge of passive movements is concerned, the muscles, quâ their contractile elements, may be entirely eliminated without detriment. I have frequently demonstrated, in cases of long standing anterior poliomyelitis, in which all the muscular tissue of a limb had disappeared so that no contraction could be elicited by the strongest galvanic currents, that the patient so affected is able blindfolded to state with accuracy whether his limb is flexed, extended or otherwise moved. Under such conditions, as is well known, not only the muscular tissue, but the anterior roots and motor nerves are completely degenerated.

It seems unnecessary to multiply arguments further. Enough has, I think, been said to show the fallacy of Brown-Séquard's doctrines in reference to the paths of the muscular sense, and the way this is affected by hemisection of the cord. Hemisection of the cord paralyses muscular sense, as well as all other forms of sensibility, on the side opposite the lesion, and leaves it unimpaired on the same side.

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[1] 'Functions of the Brain,' p. 228.

THE RELATIONS OF HYSTERIA WITH THE SCROFULOUS AND THE TUBERCULAR DIATHESIS.

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(*Continued from* Vol. VI. *p.* 460.)

II. Observations of Hysteria of a Tubercular Nature; with Pulmonary Manifestations: Reciprocal influence of the two Orders of Localisation.

Case XXVI. (condensed). (Obs. I. of the 'Iconographie de la Salpêtrière,' t. 1, p. 3.)—*Tubercular and Neuropathic Heredity. Hysteria, then Pulmonary Tuberculosis. Disappearance of the hysterical, coinciding with the apparition of the pulmonary phenomena. Death and Autopsy.*

L. Th., florist, 18 years of age. Admitted, August 1872. Father died at 42 years of age of a pleurisy; was very irritable and a drunkard; "nervous" attacks. Paternal grandfather neurotic; mother subject to bronchitis; maternal grandfather died in an apoplectic fit at 60 years of age; maternal grandmother died from phthisis, aged 46—had had twenty-one children.

During childhood, scrofulous accidents, bronchitis; of a teasing and malicious disposition. First hysterical attack in July 1871, from which time fits occur with all the typical characters: aura, hemianæsthesia, ovarian hyperæsthesia, etc.

Th. had always been chlorotic. From January 1876 had lost strength; vomiting and diarrhœa. In May, aggravation; rapid loss of flesh, with first symptoms of pulmonary phthisis, which carried her off on the 21st of July.

No hysterical attack occurred in 1876. In the beginning of May, hemianæsthesia and ovarian hyperæsthesia on the right side well marked. On June 1st hyperæsthesia less intense; pinching, pricking, cold, etc. perceived, but less acutely than on the left. Sight, smell, and taste, pretty equal on both sides, hearing less distinct on the right. July 13th: spontaneous hyperæsthesia has disappeared, ovarian pressure still painful, but no longer produced the sense of strangulation. General and special sensibility almost equal on both sides.

Autopsy: nervous system normal. Supernumerary pseudo-ovaries on the right. Adhesions at the apices of both lungs. Left lung: cavities in the upper third, granulations disseminated throughout the remainder. Right lung: several small cavities in the upper lobe; numerous tubercular masses elsewhere.

The authors of the 'Iconographie' add the following remarks: "An interesting feature of this case is the influence of tuberculosis, not upon the attacks—a well-known fact—but upon the permanent symptoms of hysteria, hemianæsthesia, and ovarian hyperæsthesia. We have seen how rapidly the latter symptoms disappear synchronously with the progress of the pulmonary lesions."

This is an important case for the view we are defending. There is a neuropathic and tubercular heredity. The diathesis is first localised in the nervous system, and is then manifested by the hysterical neurosis; next it invades the respiratory organs and determines a pulmonary tuberculosis. I look upon the hysteria and phthisis as mere successive manifestations of the hereditary diathesis, the symptoms of the one disappearing only upon the invasion of the other. An autopsy showed that there was true hysteria as well as true phthisis, and not tubercular disease of the nerve centres giving rise to hysteriform phenomena.

Such a case, observed in such a place as the Salpêtrière, will place in a better light our point of view than any theoretical discussion.

CASE XXVII. (condensed). ('Iconographie,' t. 11, p. 72.)—*Tubercular Heredity probable. Partial Epilepsy, Hysteria. Concomitant Pulmonary Phthisis, retrograding whilst the Neurosis persists.*

P. C. B., born in 1839. Admitted April 1862. Father a drunkard; died dropsical. Mother had hæmoptysis, died of bronchitis. Father and mother second cousins. Six brothers and sisters, all dead except one brother. Several attacks of bronchitis during childhood. First fit at 13 years of age; in 1860 had painful left hemiplegia. Coughs since four and a half years; had slight hæmoptysis twelve months ago. Frequent pains between shoulders and under claviculæ. Expectoration abundant, mucopurulent, somewhat fœtid since three or four months. Occasionally marked dyspnœa, and fits of coughing, especially morning and night. Percussion is painful and shows dulness on the right, subclavicular in front, supraspinous behind. Humid rales at both apices, especially on inspiration, more marked on right side. Sonorous rhonchi elsewhere. Nocturnal sweats. In 1871 the state of the limbs varied. Left leg rigid; is the seat of occasional spontaneous trepidation, which is accompanied with tonic contraction of arm and rotation of head to the left. This is the incomplete seizure. In the complete attack the phenomena are more accentuated, and combine with rhythmical clonus. Flexion of the foot determines incomplete attacks. The patient sometimes passes whole days in a state of ecstasy, with occasional tremors of the limbs, but no general convulsion.

In 1872 there occur seizures in which loss of consciousness is not complete, the eyes closing upon the approach of the finger. On one occasion no fit, but peculiar state of abstraction; hallucinations of ear and eye.

In 1873 phenomena of the kind described as "hysteriform agitation" are described, reaching such an intensity as to necessitate recourse to the strait-waistcoat. After one of the attacks, patient is seized with laughter, stating that she hears what is said but does not understand it well; after another, during which the eyelids moved rigidly, a persistent contraction of the left leg ceases rapidly and sets in again an hour after. The patient also had convulsive attacks without cry or loss of consciousness, involuntary evacuation, nor foaming at the mouth, nor any consecutive stupor. Towards the end of the year distinct series of hysteriform fits.

From 1875 to, 1877 similar condition persists. Foolish and erotic delirium, then true "status hystericus." Hysterical and epileptic manifestations alternate with one another.

In June 1878 the author writes: "For a long time we have had no particular accidents to note with reference to the pulmonary organs. In the right apex, back and front, slight diminution of resonance, rough vesicular murmur, weaker than in the left. Coughs rarely." We need not give here a fuller account of the nervous symptoms, for which the reader is referred to the original.

This case is almost as remarkable as the preceding, although from a different point of view.

First it will be noticed that I place it under the heading of

Hysteria, although in the 'Iconography' it goes by the name of partial epilepsy. The details to which I have drawn special attention in the foregoing epitome, show that by the side of the partial epilepsy there really was a hysterical element in the patient. I should even be inclined to look upon the partial epilepsy as of hysterical origin, now that I have read the last note of Ballet and Crespin ('Progrès Médical,' Feb. 2nd, 1884) on the attacks of hysteria simulating partial epilepsy. But whatever view we take of it, the case can be utilised here.

It will be noticed that the tubercular heredity showed itself almost simultaneously in our patient by pulmonary symptoms and neurotic phenomena. Only it is a remarkable fact that instead of hysteria yielding to pulmonary tuberculosis, as was the case in the preceding observation, it is the phthisis which has given way to the hystero-epilepsy. For it is enough to compare the two examinations of the chest made in 1866 and in 1878, to see the enormous amelioration which has taken place in the phthisis which was already serious at the time of the first examination, and had been but very slightly treated in the interval. It is a pity that repeated examinations were not made during those twelve years, so as to show us the progress of the pulmonary lesions; but it does not prevent this observation from being of considerable interest.

Case XXVIII. (Obs. IV. of Fouquet. Thesis; Paris, 1880, 343; p. 28.)—*Neuropathic and diathesic heredity (rheumatic and tubercular). Hysteria; stercoraceous vomiting, convulsions. Pulmonary tuberculisation—alternations between the nervous and thoracic phenomena.*

Heredity.—"Her father's death was caused by an accident. Her mother is very nervous. One of her sisters died phthisical at 18. Another is in pretty good health just now, but has had two attacks of acute articular rheumatism. This last sister is married, and has had two children who died of convulsions. She has also two brothers, both in good health."

History of the patient.—20 years of age; face yellowish, pale; characteristic aspect of chlorosis. Very nervous, laughs and cries without motives. Hysterical fit with stercoraceous vomiting. Very evident crepitations at the apex of the right lung. Later on, convulsive epilepsy with left ovaritis and hemianæsthesia.

"A fact worthy of notice is that when her attacks came back, the cough was less troublesome and the state of the lungs seemed to improve; but as the patient was obliged to interrupt her work, she then took bromide of potassium; then, as the nervous accidents diminished in intensity, the state of the lungs grew worse again. In 1876 the two lungs were attacked, and in April 1877 the patient succumbed. She had not had any hysterical accidents during the last six months of her life."

Notwithstanding the absence of details, this summary brings into relief the same principles which were illustrated in Cases XXVI. and XXVII. We see here a very curious and repeated alternation between the hysterical and thoracic manifestations of the hereditary tubercular diathesis.

The author expresses himself clearly on the amelioration of the lung when the hysteria predominated. He shows the successive phases of the struggle, and the final victory of the pulmonary tuberculosis, which destroyed the patient after having altogether silenced the neurosis during the last six months of life.

CASE XXIX. (condensed). (Obs. VII. of Briquet, loc. cit. p. 29.) —*Man; Neuropathic Heredity (father and sister), but doubtful as to the tuberculosis (mother). Hysteria; Tuberculosis. Death in Marasmus; tubercular granulations in apices of lungs.*

The mother, whose health was delicate, died in early life, of a disease which may have been either a nervous affection or a cancer of the stomach. The father died when 55 years of age, after two months of mental alienation with incipient paralysis of the lower limbs. His sister suffers from a nervous affection.

Always impressionable and easily moved; he has from 25 to 32 years of age been exposed to much worry. At that time various nervous phenomena appear. When 35 years old, convulsive attacks, leaving after them weakness and numbness in the left side of the body, then permanent pain in the middle of the back. He is admitted at "La Charité" when 37, in 1851.

The author describes the hysterical phenomena; then goes on as follows: "The thorax is about normal; resonance deficient under the right clavicle, and the vesicular sound is a little weak in that spot. From time to time there occur attacks of dyspnœa. No cough, no expectoration. When he exerts himself to cough, to swallow or to move about freely, one hears during inspiration a kind of stridor, resembling that of the first stage of a fit of whooping-cough; suffocation seems imminent. At that time very frequent convulsive attacks, anæsthesia, etc.

"Gradually the nightly attacks become less frequent, but the patient got weaker and lost flesh notably. The difficulty experienced in the act of deglutition increased, the ingestion of food became more and more difficult. Gradually the respirations became embarrassed, the laryngeal sound increased, and at the same time the weakness rendered the cough, the expectoration, and the breathing more and more difficult. The continual epigastric pains and the nocturnal attacks have given rise to a feverish condition. Anæsthesia and paralysis have come back again more marked than before. The intellect, however, has always been wholly unimpaired. Death happened on May the 4th, after four months in hospital."

At the autopsy, nothing was found in the nervous centres except a little adhesion of the arachnoid and the pia-mater (thickened but transparent) to the brain.

"The lungs contain at the apices some miliary tubercular granulations." Nothing to note in other organs, except a slight thickening, of a lardaceous character, of the pylorus without noticeable stenosis."

We find again here, as in the three preceding observations, the coexistence in the same subject of hysterical and pulmonary manifestations. Here, too, the pulmonary tuberculosis progresses but slightly, and remains at the preliminary stage as in Case XXVII., the hysterical phenomena (at least the permanent phenomena) persisting, on the contrary, with great intensity.

This case differs from the preceding, however, inasmuch as the diathesis has killed the patient, whilst remaining but slightly marked in its thoracic localisations. The patient of Observation XXVI. died with the lesion of an advanced pulmonary phthisis; this one, on the contrary, with almost insignificant pulmonary mischief: he has succumbed to his general state, but not to the localisations of the diathesis.

Thus, in this patient, the constitutional affection has manifested itself both by the hysterical phenomena and by the thoracic symptoms. Owing to this double localisation, and to the predominance of the neurosis, the pulmonary manifestations have remained very slightly accentuated. But the progress of the diathesis has nevertheless continued, the general nutrition has been altered, and the patient has succumbed in marasmus, thus plainly showing that the evolution of the tubercular diathesis is not necessarily parallel with the evolution of pulmonary tubercles.

Case XXX. (condensed). (Obs. VI. of the Thesis of Chambard, p. 120.)—*Tubercular and Neuropathic Heredity. Pulmonary Tuberculosis; afterwards Hysteria.*

Father violent, alcoholic, and a libertine. Mother very nervous, dead of cancer of uterus. Many relations alcoholic. Brother dead of disease of the chest. Other brother, 20 years old, always ill, coughs much. Sister dead of disease of the chest.

Two years before her admission into the hospital (May 3rd, 1879), after a violent access of anger, pigmentation of face, which disappears rapidly with the application of tincture of iodine, but returns permanently four or five months after. Six months before, right pleurisy, which was rapidly cured.

"A month ago, following upon a chill, shivers and feverishness occurred, which kept the patient in bed four days; then she began to cough, and had two copious hæmoptyses. She also complains of profuse perspirations during the night, of vesperal feverish attacks and of loss of flesh; in a word, of the majority of the general signs of the pulmonary phthisis which the exploration of the lungs allows to detect easily." "It is because of these general disturbances brought on by that disease that she seeks admission to the hospital." "The expectoration is almost absent. The right apex shows evident signs of pulmonary tuberculosis. Harsh respiration, vocal resonance increased, rhonchus and lacunar crepitations. The same signs are present at the left apex, but less marked."

The report contains a detailed study of the hysterical phenomena observed or provoked in the patient. Unfortunately there is no information upon the ulterior evolution of the disease.

What is most interesting in this new example of coexistence of thoracic and hysterical signs in the same patient, is the antecedence of the pulmonary symptoms, whilst in the preceding case the neurosis occurred before the respiratory tuberculosis. Unfortunately this observation proves but little: first, because the hysteria is almost altogether a provoked one; and secondly, because no details are given concerning the ulterior history of the subject, and the respective evolution of the various symptoms.

CASE XXXI. (Largaud, Thesis of Montpellier, 1882, No. 23, p. 22.) —*Pulmonary Tuberculosis. Hydrotherapy. Hysteria. Disappearance of the pulmonary phenomena. Cure.*

"Miss X., age 20, was for two years under the care of Dr. Masson and Dr. Cavaillon for frequent hæmoptyses, with obstinate cough, intercostal pains, decline, and dyspeptic troubles. The auscultation revealed crepitations at the apices, which caused the doctors in attendance to diagnose incipient pulmonary phthisis. The ordinary treatment completely failed. Dr. Masson then ordered cold lotions to the extremities against the recurrent hæmoptysis, which ceased after a few days. The general state of the patient remaining always as bad, and the phthisis seeming to advance, Miss X. was submitted to a hydrotherapic course. After a shower-bath, a formidable hysterical fit broke out. A true neuropathic state manifested itself, and lasted a whole year, during which the morbid pulmonary phenomena improved in a marked way. A new affection then appeared which cut short the hysterical neurosis in the shape of a sciatica, which for eight months resisted every treatment. A furuncular eruption brought on a recovery from the sciatica, and the patient no longer showed anything special with reference either to the lungs or nervous system."

The succession of the symptoms here is very curious. Tubercular lesions in the lungs, then hysteria; the first symptoms disappear when the second begin. The sciatica takes the place of the hysteria, the boils take that of the sciatica, and lastly everything gets well. It is very difficult to look upon these as a series of distinct and antagonistic diseases, succeeding each other in the same subject. It is much more natural, and according to clinical experience, to look upon all these facts as being symptomatic manifestations, different and successive, of one morbid factor, the tubercular diathesis.

Let us notice finally the fact of the cold shower-bath bringing on the first attack of hysteria.

CASE XXXII. (Largaud, loc. cit. p. 30.)—*Pulmonary Tuberculosis. Hysteria. Succession of the two orders of manifestations.*

"During the year 1874, the woman B., 30 years of age, has spent 6 months at the hospital of Carpentras, with all the signs of a softening at the apices of the lungs (hæmoptysis, cough, abundant nocturnal sweating, loss of flesh, dyspeptic troubles). She suffers afterwards of a series of most violent hysterical fits, and, during that time, the morbid pulmonary state improves from day to day. She leaves the hospital. Two years later, the woman B. has a child; she does not cough at all, and the most careful auscultation is negative. This woman has just had a fit of lumbo-abdominal neuralgia of indescribable violence coming after a chill, and simulating a myelitis of the lumbar region. She is pretty well to-day."

Given more in detail, this case (as well as the next) might prove interesting; epitomised as it is, we give it as simply indicating one fact to compare with the others brought together in this paper.

CASE XXXIII. (Largaud, loc. cit. p. 23.)—*Pulmonary Tuberculosis, then Hysteria.*

"The girl X., from 1871 till 1878, has spent three or four months of every winter in the hospital of Carpentras. She has had, first of all, many hæmoptyses, and presented sure signs of softening at the apices. The hysteria showed itself some time after (slight convulsions, globus, hiccough, etc.), since then, our patient, whose phthisis has improved, still comes back to the hospital every winter, showing signs of a general neuropathy, consisting in spasms, in a fidgety and talkative disposition, and in various visceralgias. She is always pale, thin, and has a characteristic dry and fitful cough. Auscultation, however, reveals nothing. She has a special appearance (vague look, dilated pupils, and tendency to ecstatic attitude). The girl X. is still alive to-day."

CASE XXXIV. (Largaud, loc. cit. p. 24.)—*Pulmonary Tuberculosis, then Hysteria.*

"The girl M. has at first presented all the symptoms of a pulmonary tuberculosis progressing slowly (cough, hæmoptyses, considerable loss of flesh, nocturnal sweating). Seized with a violent hysterical fit, followed by many others, the phthisis improved and disappeared, giving place to a hysterical paraplegia, with prolapsus recti, which for five years resisted all treatment. The cure of her paraplegia has made a great sensation, for it is, she says, after nine days' prayers at 'Notre Dame de Santé,' that she has been cured. In the month of July 1880, she wanted an operation to be performed for the prolapsus recti. After resection,

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peritonitis set in, and she died four days after. The autopsy could not be made."

CASE XXXV. (Largaud, loc. cit. p. 25.)—*Pulmonary Phthisis and Hysteria. Alternating predominance of the two orders of symptoms. Tubercular Cachexia; Diarrhœa; Death.*

Philomène S. remained one year in the hospital of Carpentras, for nervous attacks.

"Patient is a foundling; she is twenty; during the time of her youth she has been pale, puny, and has presented sure signs of scrofula (ganglionary swellings of neck, very obstinate eruption on the head, etc.).

"During her one year's stay at the hospital, she has presented alternations of obstinate cough (we cannot here define the state of the lungs) and hysterical fits. These were either powerful attacks with globus and convulsive movements, or mere adumbrations of fits. The whole course was very irregular. She left the hospital very much better, as far as the cough and the neurosis were concerned, but her general state was still very bad.

"Philomène married some time after, and gave birth to a child which only lived a few days.

"She was lost sight of up to the day when she was admitted into the hospital for an obstinate diarrhœa (January 3rd, 1880).

"She says that since her confinement she has had a few fits, which have diminished in number and in intensity from the time when the cough has come back stronger, and the diarrhœa manifested itself.

"She is very much thinner, very pale, and her courses have stopped. The auscultation reveals crepitation at both apices, especially on the right side, and prolonged expiration. At night, when the respite given her by the cough allows her to sleep a little, she is at once bathed in perspiration. She has as many as ten motions a day, especially in the morning, and the fæces present the appearance of clay, and are of the consistence of pap. They are almost odourless, and contain neither shreds of false membranes nor any traces of blood. As to the neurosis, its only manifestation is a certain mental alternative. She chatters incessantly, tells lies at every opportunity, and passes without reason from laughter into sobbing fits. Not the slightest appearance of convulsive attacks.

"The hysteria remains hardly noticeable for three months, and would perhaps have been overlooked, but for the previous history; but auscultation and expectoration give us daily fresh evidence of the progress of the phthisis, so much so, that from day to day a fatal termination is apprehended.

"On April 10th, we are called at midnight. The following is the state in which we find her: She is shaken by movements so inordinate that all the nurses can do is to keep her in bed. She cannot speak, but she puts one of her hands to her throat, and seems to beg that the assistants would tear away something which grasps and chokes her. The eyes are haggard, respirations noisy and stertorous. Then begin clonic convulsions which last for about a quarter of an hour, after which the patient, exhausted, falls to sleep till the morning.

"There we have evidently a return of the hysteria with a regular attack. From that day she has nothing but fits declining in intensity (8 to 10 in number), and we notice with astonishment that all the phthisical symptoms are daily improving. On the 1st of September, the lungs have nearly recovered their natural action, and the patient has regained a little stoutness and strength; although the diarrhœa persists (3 to 6 motions in 24 hours), resisting all the treatments directed against it.

"In the month of March of the following year, Philomène leaves the hospital, still with her diarrhœa, and goes back to her occupations.

"We have since heard that the patient, again considerably weakened by the diarrhœa, has once more been admitted into the hospital during the first days of January 1882. Her hysterical attacks had completely ceased, and the diarrhœa had given place to a generalised œdema to which she succumbed on the 1st of March, without other characteristic symptoms of tuberculosis.

"We regret that the autopsy could not be made."

What strikes one in this case, as Dr. Largaud says, "is the slowness with which the symptoms of phthisis have progressed in a patient so evidently affected by the tubercular diathesis.

"At first both phthisis and neurosis progressed together, a kind of equilibrium being produced between the two affections. Later on, the pulmonary phthisis manifested itself in a formidable outburst. It then stopped almost suddenly, and at the same time the hysterical attacks of the greatest intensity began.

"The thoracic accidents then gave place to an intractable diarrhœa, evidently tubercular in character, and which in the end carried off the patient."

CASE XXXVI. (Largaud, loc. cit. p. 28.)—*Pulmonary Phthisis at the beginning; Hysteria displacing the thoracic symptoms.*

Marie X., age 27, admitted into the hospital of Carpentras the 3rd of February, 1881. Cannot give any information concerning her heredity. Seems to have had syphilis when 21 years old; but no trace of it is found at the time of her admission except a chronic laryngitis with very harsh voice. Rheumatic pains (?) for last two months. She has taken a violent cold, degenerating "into an acute bronchitis, which assumes from day to day more the tubercular character; first sanguineous expectoration, then veritable hæmoptysis; abundant nocturnal sweatings; noticeable weakening and loss of flesh. The auscultation reveals dry and very harsh crepitations at both apices, and prolonged expiration. Percussion yields a great difference of resonance between the apices and the bases, especially on the right side. Fever."

"Patient was growing daily worse, when about middle of April she was suddenly taken, at two o'clock, P.M., with a violent hysterical fit. She was near the stove when she felt herself getting weaker, to such an extent that she had to be carried to her bed. She then experienced in the epigastric region an abnormal weight, as if something were rising, clutching her at the throat and choking her. She is extremely agitated, screaming aloud, then falling as if she were lifeless. Convulsions supervened, consisting of pretty regular alternations of contraction and of flexion of the legs and arms, then movements of pelvis ('cynical phasis'). After a few moments, the patient, exhausted, falls asleep.

"The next day, a second fit at the same time; then a third and a fourth one, all identical. Since that time the state of our patient has altogether altered. The pulmonary affection improves very rapidly, so much so, that after a month every trace of phthisis seems to have disappeared."

"The hysteria goes on, but is of a benignant type (mind, neuralgia, anæsthesia, etc.), and the patient leaves the hospital in September, keeping nothing else of her neurosis than certain mental peculiarities."

This observation has not perhaps the importance given it by Dr. Largaud, because the pulmonary phthisis is insufficiently proved. Nevertheless we see that instructive succession of phenomena: thoracic troubles exhibiting the signs of an incipient pulmonary phthisis, the place of which is afterwards completely taken by a marked hysteria which sets in suddenly.

Case XXXVII. (Largaud, loc. cit. p. 81.)—*Incipient Pulmonary Tuberculosis, replaced afterwards by Hysteria.*

Louise B., 17 years old, "assistée" child of the Hospice, is admitted into the hospital of Carpentras on March 4th, 1881, with all the symptoms of chloroanæmia. Douches and ferruginous preparations. After sixteen days of this treatment, she is taken with violent cough and sanguineous expectoration; at the time of admission auscultation had revealed nothing. Notwithstanding the suppression of treatment the patient coughs more and more, and crepitations are heard in the whole apex of the right lung, as well as harshness of the respiratory sounds, and prolonged expiration on both sides. Hæmoptyses, nocturnal sweatings. The patient, very much weakened, can no longer leave her bed. The diagnosis made is tubercular bronchitis.

Things grow worse till the 1st of May, at which time the patient is suddenly taken, during the morning visit, with slight convulsions, followed by deep prostration. On the same day, at 8 and 10 in the evening, other fits like the first.

The next morning at the time of the visit, aphonia, which persists with alternations. Slight convulsive fits every day, and constriction of the throat, anæsthesic and hyperæsthesic regions. This condition persists for two weeks, and at the same time there is a marked improvement in the respiratory system, and the general state. Then about the middle of June, the hysterical phenomena improve in their turn, and on July the 1st, Louise B. seems quite cured, and asks to leave the hospital. She goes out on the 1st of September, at which time the symptoms of pulmonary phthisis have disappeared, and of the neurosis there only remains a great tendency to lying and chattering.

The author sees the patient again in March 1882, and finds nothing in the lungs, but during the last month she shows fresh symptoms of the neurosis. She suffers every other day from formidable hysterical fits, with globus, tonic and clonic convulsions, erotic in character, and with loss of consciousness. The compression of one of the ovaries calms the fit almost instantly, whilst the compression of the other increases its intensity, and draws loud screams from the patient.

With the same reservations that we have made concerning the preceding observation, we have here again a remarkable example of thoracic phenomena, probably tubercular, replaced later on by hysterical manifestations. Let us add that the rapid disappearance of the latter was only an apparent recovery, and that the hysteria had really taken possession of the patient with its usual characters of permanence and tenacity.

CASE XXXVIII. (Largaud, loc. cit. p. 86.)—*Tubercular Heredity; Pulmonary Tuberculosis; disappearance of those phenomena and appearance of fits of Somnambulism.*

Rose C., 7 years of age. Father and mother dead in 1879 of pulmonary tuberculosis. She, after a cold, becomes feverish, has an obstinate cough, slight hæmoptyses, nocturnal sweatings; loses flesh exceedingly rapidly. At first crepitations, then evident signs of softening in the right apex. Three times these symptoms abate, and then reappear. At last, eight months after the beginning, marked and lasting amelioration. There only remained, to the great astonishment of the doctors, slight neuralgia in the intercostal spaces and in the legs.

"One evening the sister calls us to show us Rose in a fit of somnambulism, walking in the ward with her eyes shut, and stopping at every bed to fill the patient's glasses with medicine; she goes by us without seeing us, and goes back to bed without saying a word. The sister informs us that similar somnambulistic attacks had already been noticed of late." Rose leaves the hospital in November 1880, in a very satisfactory state.

Here the pulmonary tuberculosis is hereditary, it begins with unusual intensity for a child 7 years old. Afterwards she recovers against all hopes, and fits of somnambulism appear.

"We think," Dr. Largaud says, "that the cure of the phthisis in this child of 7, phthisis which seemed likely to go through its different stages in a very limited time, is much less due to the treatment followed, than to the appearance of a neurosis, which at first has checked and afterwards removed the thoracic accidents."

We reserve the theory of Dr. Largaud for further consideration, but the fact itself is very important as far as our argument is concerned.

CASE XXXIX. (condensed). (Berdinel, 'Arch. de Médec.' 1875. Chataing, Thesis, Paris, 1880, No. 213.)—*Heredity unknown. Hysteria. Beginning of Pulmonary Tuberculosis which does not progress.* (*The observation includes a space of* 8 *years.*)

Lecomte (Marie Elisabeth), 29 years of age. Admitted into the hospital Cochin, the 29th of May, 1873.

Foundling. No previous history, except a typhoid fever in 1870 or thereabout. In April 1873, right pneumonia. Persistent anuria after the application of blisters. In June, symptoms of pelvi-peritonitis. The general state is disturbed, and at the same time the patient becomes whimsical, etc. The vague nervous troubles accentuate themselves. Pains in abdomen; vomiting. In November, sleep of three days. The nervous phenomena become more marked. The anuria persists.

In January 1874, left hemianæsthesia. Anuria and vomitings. Recurrence of pelvi-peritonitis. In June paralysis of left leg. On the 16th of August, beginning of pleurisy on the left side with effusion. In October, anæsthesia and paralysis become paraplegic. Sometimes in the beginning of December, a few fits of coughing, followed by slight hæmoptyses accompanied by thoracic pains. However, anuria and paraplegia persist.

1*st January*, 1875.—Patient has a tired look, is thin; she has coughed a little for some time back, and has had some hæmoptyses; auscultation reveals at the right apex a few dry crepitations; still paraplegia with anæsthesia. Voice harsh, almost toneless for more than three months. Vomiting and ischuria. The hysterical state shows itself also by a marked mental peculiarity, by diffuse neuralgia, by feelings of suffocation which, for some weeks past, have taken the characters of the usual globus; profound anæsthesia of the ocular and pharyngeal mucous membranes. Semicataleptic sleep in March. Later on, pulmonary congestion of the left side, with fever. In April, long fits of sleep for 17 days. (See the very carefully analysed details in the work of M. Berdinel.) The patient leaves the hospital on July 29th, and M. Berdinel adds: We have since seen the patient; she does her work, which however is not trying, fairly well, but she shows signs of pulmonary tuberculosis. Her voice is rather hoarse, and she vomits from time to time after food.

The patient does well for four years, except for an attack of erysipelas of the face. On March 10th, 1880, she is taken with pains in the throat and with aphonia, then the urinary troubles reappear; vomitings, bilateral ovaritis, right hemianæsthesia.

Sensory disturbances. In the midst of the history of the progress of those nervous phenomena we find (April 28): The patient looks well, but she always perspires very freely. She complains of a slight cough, but auscultation reveals nothing but a little respiratory harshness at the apices. On May the 13th, she leaves the hospital, suffering only from aphonia.

In that celebrated hysterical patient, whose fits of sleep at the hospital Cochin have created a great sensation (see, besides the paper of Berdinel quoted previously, that of Monod in the same journal, 1877), the thoracic and neurotic manifestations exist together and complicate one another. Alternation is therefore not observed in her case, the two orders of symptoms do not take each other's place. Only the tubercular lesions, instead of going forward and progressing, remain stationary, and even retrograde during that long period of eight years, during which she is closely observed, and during which the various manifestations of the neurosis predominate greatly.

This observation differs from the majority of the preceding in certain particulars; but it is connected with them in other aspects, and establishes at any rate a clinical type worth remembering.

(*To be concluded.*)

ON THE CONDITION OF THE FUNDUS OCULI IN INSANE INDIVIDUALS.

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SINCE the introduction of the ophthalmoscope, the investigation of the condition of the fundus oculi in insane individuals has occupied the attention of many observers; though in this country, at least, the published observations are not very numerous. It cannot be said, however, that the subject is one which is yet by any means fully elucidated; indeed, the discrepancies between the recorded observations of different investigators is so great, that to reconcile the opposing statements is no easy task. With the view of endeavouring to throw some light on the question, the investigation shortly to be described was undertaken, and though we are sensible that the number of observations is too small to admit of anything like a settlement of the points in dispute, we think that a record of them will not be without its value.

It need hardly be said that in many insane individuals, the use of the ophthalmoscope is attended with much difficulty; some patients give trouble by their restlessness and turbulence and by the impossibility of fixing their attention; others again become much alarmed when they are brought to the "dark room," and no persuasion or assurance will get them to submit to an examination. By taking care however, that the pupils are thoroughly dilated with atropine, and by the use of patience and perseverance, these difficulties can in the great majority of cases be overcome. We have, however, in a few instances, been compelled to abandon the examination as altogether impracticable, and in a few other cases, only an imperfect view being obtained, we could not feel certain of the result; all such cases are however excluded from the descriptions which follow, which deal only with those about which we feel that we can write in full confidence.

Our observations have been conducted with the most recent refracting ophthalmoscopes, the "indirect" method having been in almost every case supplemented by the "direct;" we have also, in the majority of cases, taken care to note such errors of refraction as existed, and have endeavoured to measure the amount of these as closely as possible. In the case of General Paralysis we have further supplemented the investigation by the use, wherever practicable, of test types, with which we have obtained reliable results in a much larger proportion of cases than we anticipated when we commenced. It may indeed be objected by some, that subjective examination in persons mentally alienated can never give results of scientific value, but this objection applies only to the negative aspect of the question; if a lunatic *reads* correctly certain test types it is obvious that he can *see* them, whereas the failure to read them wholly or in part, does not necessarily imply inability to *see* them, since his incapacity in this respect may be due to lack of attention, defect of mental power, or other cause. We should have thought indeed that the careful use of test types as a supplementary aid in an investigation of the kind before us scarcely required defence, were it not that no less an authority than Dr. Clifford Allbutt has stated[1] "that failure of sight as tested by types is wholly an oculist's symptom, and bears little proportion to the amount of atrophic or other disease that may be seen in the nerve." It is indeed well known that a patient's sight may be perfectly intact, even when intense papillitis is disclosed by the ophthalmoscope, the explanation being that the conductivity of the nerve tubules is as yet unimpaired; and we quite admit that it is often impossible, from a mere inspection of an atrophied disc, to pronounce definitely as to a patient's acuity of vision, but we think that the cases of optic nerve atrophy have yet to be described in which a patient is capable of reading J 1 and $\frac{20}{20}$, and in a doubtful case where the diagnosis lies between a slightly atrophied and an anæmic optic disc, we think the use of test types may often give valuable help. And this leads us to make a few remarks on the normal colour of the disc; it can, we think, hardly be doubted, that the earlier observers took too rigid a view as to the physiological differences in colour here displayed; it was not fully recognised, and is even now, we think hardly sufficiently so, that the colour of the disc may vary in different individuals from an almost uniform redness to an almost uniform pallor, and yet the disc in question may be in a perfectly normal condition. We have an instructive case under observation now, in the person of a female epileptic whose discs are of an almost uniform whiteness strongly suggestive of atrophy, and yet the patient can read *perfectly* J 1 and $\frac{20}{20}$. In fact we cordially endorse the statement made by Dr. Gowers,[1] "that the tint of the optic discs may vary as much as the tint of the cheek."

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[1] 'The Ophthalmoscope,' p. 380.

We wish it had been possible to have in addition tested the field of vision, but we do not consider that reliable results could be obtained by this method in the persons of lunatics.

In some of the cases of General Paralysis, we have made a microscopical examination of the optic nerves and discs after death, but we have not thought it necessary to insert any account of the naked eye appearances of these parts, as we consider that these appearances furnish *usually* but a very rough guide to the intimate structure, and are therefore not to be relied upon.

Our observations have all been made in the wards of Rainhill Asylum, and we are indebted to Dr. Rogers, Medical Superintendent of that Institution, for the facilities afforded us in the examination of patients. The investigation has extended over a period of two and a-half years, and has embraced cases exhibiting every variety of mental disorder. The results will be given in detail as we proceed, but we may say at once that the actual lesions noted have been comparatively few.

A word or two first, however, with respect to the classification of mental disorders here adopted. For a purpose such as that before us, we think that the simpler we can make this, the better, and the old division of Insanity into Mania, Melancholia and Dementia, has at least the advantage of simplicity, and the further one of being in possession of the field: many varieties of mental disorder are, however, included under each of these heads which cannot in strictness be grouped together, and therefore, whilst retaining it as our basis we have elaborated it in detail. In the first place then, we divide all our cases into General Paralysis, and Not-General Paralysis; and the latter class we subdivide as follows: of Mania we make four subdivisions; "Acute" including all recent cases slight and severe; "Puerperal," which is of course simply a variety of acute, but to which it is perhaps better to assign a separate heading; "Chronic"; and the "delusional form," which includes the cases commonly known as "Monomania." Of Melancholia we make a threefold division, into "active recent," "passive recent," and "passive chronic."

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[1] 'Medical Ophthalmoscopy,' 2nd edition, p. 4.

Cases of Mental Stupor we include in a class by themselves. Cases of Dementia are subdivided according as they are "Primary" (including what is known as Acute Dementia whether recent or chronic), or secondary; the latter again into cases secondary to Mania and those due to gross organic disease of the brain, such as old Cysts, Hæmorrhages, &c., and which may be styled "organic;" whilst a fourth subdivision comprises all "senile" cases, *i.e.* cases of primary dementia in old people not known to be due to gross changes.

We make a separate division of Epilepsy, which again we subdivide according as it is associated with imbecility—with slight—or with grave—impairment of the mental faculties. Cases of Imbecility, uncomplicated with Epilepsy, are also included under a separate heading.

Our classification will then stand thus:—

I.—General Paralysis.

II.—Not-General Paralysis
- 1. Mania . .
  - *a.* Acute.
  - *b.* Puerperal.
  - *c.* Chronic.
  - *d.* Delusional form of.
- 2. Melancholia
  - *a.* Active, recent.
  - *b.* Passive, recent.
  - *c.* Passive, chronic.
- 3. Mental Stupor.
- 4. Dementia .
  - *a.* Primary (recent and chronic)
  - *b.* Secondary to mania.
  - *c.* Organic.
  - *d.* Senile.
- 5. Epilepsy .
  - *a.* With imbecility.
  - *b.* With slight dementia.
  - *c.* With grave dementia.
- 6. Imbecility (uncomplicated with epilepsy).

Taking all forms of Insanity together, we have examined 313 different cases (persons); of these 66 were General Paralytics, and 247 not-Paralytics; of these latter in 204 (82·59 per cent.) the optic discs, and main retinal vessels were perfectly normal, whilst in the remaining 43 (17·40 per cent.) these parts were either abnormal or were marked as doubtful, *i.e.*, in these latter the changes were so slight, that no decided opinion as to their abnormality could be formed. In some of the abnormal cases again, the changes noted (as will be seen in detail as we proceed) were clearly due to causes such as Bright's disease, which had no connection with the patient's lunacy or at best but an indirect one; and these cases must of course be allowed for, in endeavouring to trace a connection between the patient's mental (cerebral) state, and the condition of his optic discs or retinal circulation. Furthermore, as is well known, a variety of functional constitutional states are at times associated with changes in the discs, altogether apart from the question of lunacy. The effects of errors of refraction have also not unfrequently to be discounted. If due regard be paid to these considerations it will be found that the percentage of cases in which we have noted a distinctly abnormal condition of the discs, &c., in connection with mental alienation (other than that associated with General Paralysis) is a very small one.

We will now proceed to give in detail the observations we have made under each head; after which we purpose briefly to refer to the more important communications on the subject that have appeared in this country, and finally to draw some conclusions.

We will begin with cases of General Paralysis, this affection being not only the most important, but that in which we found by far the largest proportion of departures from the normal condition.

[EDITORIAL NOTE.—Circumstances independent of our own will have compelled us to let the main portion of this paper, and the Plates which illustrate it, stand over till our next issue. The following Tables contain a synopsis of the cases referred to by the Authors.]

TABLE I.—Showing the Condition of the Eye

No.	Initials of Patient.	Sex.	Age.	Stage of Disease.	Dates of Examinations.		Condition of Fundus, &c.
1	E. McM.	F.	24	1st	'81, Oct.	29	Blurring of inner edge of each disc (mo marked in left).
				2nd	'82, Mar.	22	Same condition.
				,,	,, Nov.	4	,, ,,
				,,	'84, Feb.	2	Condition absolutely unchanged.
2	H. B.	F.	44	1st	'81, Nov.	7	Both discs normal; arteries very small, an few in number.
				,,	'82, Mar.	15	Both discs pale.
				,,	,, ,,	29	,, ,,
				2nd	,, June	10	,, ,,
				3rd	'83, Jan.	25	,, ,,
3	A. B.	F.	56	2nd	'81, Nov.	2	Both discs perfectly normal.
				,,	'82, Mar.	15	,, ,, ,, (pink).
				,,	,, Sept.	14	Both discs very red, nearly the colour of th fundus; redness invades centres; margin of left disc decidedly blurred.
				,,	,, Nov.	23	Same condition.
4	A. R.	F.	38	1st	'81, Nov.	2	Both discs perfectly normal (red). Large st phylomata around each, developed chiefl to inner sides.
				,,	'82, Mar.	15	Same condition.
				,,	,, ,,	29	,, ,,
				2nd	'84, Feb.	2	,, ,,
5	A. W.	M.	42	2nd	'81, Nov.	5	Both discs perfectly normal.*
6	E. L.	F.	33	2nd	'81, Oct.	16	Both discs perfectly normal. (Indirect ex amination only.)
7	T. C.	M.	41	2nd	'81, Nov.	9	Both discs perfectly normal.
				,,	'82, Mar.	11	Same condition.
8	J. O.	M.	37	2nd	'81, Nov.	10	Both discs perfectly normal. (Periphera striæ in both lenses.)
				,,	'82, Mar.	11	Same condition.
9	T. P.	M.	32	2nd	'81, Nov.	10	Right disc perfectly normal. (Left eye—opaque capsule.)
				,,	'82, Mar.	11	Right disc perfectly normal.
				,,	'84, Feb.	20	,, ,, ,,
10	J. C.	M.	43	2nd	'81, Nov.	10	Both discs perfectly normal. Numerous smal pigment spots in right retina.
				,,	'82, Mar.	11	Same condition.
11	F. W. T.	M.	43	2nd	'81, Dec.	17	Both discs perfectly normal. A small patc of choroidal atrophy in vicinity of righ macula.
					'83, Jan.	5	Same condition.
12	E. D.	F.	21	2nd	'81, Dec.	7	Both discs perfectly normal. (No staphy lomata.)
13	C. B.	F.	42	1st	'81, Dec.	3	Both discs decidedly red, the area of greate redness being a middle zone between cent and margins, which are pale.
				,,	'82, Feb.	1	Both discs very red, almost the colour of th

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* In this and all other cases where no special reference is made to the conditi

TY-SIX CASES OF GENERAL PARALYSIS OF THE INSANE.

Refraction of Eyes.	Acuteness of Vision.	Condition of Pupils.	Termination of Case.	Remarks.
R. + 3 D. L. + 2 D.	R. $\frac{20}{40}$ J. 1 L. $\frac{20}{30}$ J. 1	3½ mm. equal, regular; act well to light and to accommodation.	Still under observation.	The blurring of inner edge of each disc is probably a physiological condition.
mmetropic	..	R. 5 mm., L. 3. regular; very little action either to light or to accommodation.	Died, '83, July 26.	Had aortic regurgitation.
R. + 1 D. L. + 1 D.	R. $\frac{20}{40}$ J. 16 L. $\frac{20}{30}$ J. 16	R. 3 mm., L. 4 mm., both regular; sluggish to light, but act to accommodation.	Died, '83, Jan. 20.	Died of phthisis. Microscopical examination of optic nerves, &c. Pachymeningitis of cerebral dura mater.
R. – 10 D. L. – 10 D.	R. $\frac{20}{0}$ J. 2 L. $\frac{20}{0}$ J. 2	R. 3 mm., L. 4 mm., regular; act to light and to accommodation.	Still under observation.	
..	..	.. ..	Died, '81, Nov. 30.	Died of an apoplectiform attack.
Emm.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{30}$ J. 1	Both 2½ mm., act to light, but very sluggish.	Died, '81, Oct. 31.	Died of diarrhœa.
Emm.	R. $\frac{20}{30}$ J. 1 L. $\frac{20}{15}$ J. 1	R. 3½ mm., L. 3 mm.; right very sluggish to light, left acts well; both act to accommodation.	Died, '82, Dec. 14.	
R. + 2 D. L. + 2 D.	R. $\frac{20}{40}$ J. 2 L. $\frac{20}{40}$ J. 4	Both 3 mm., regular; no action to light, but act well to accommodation.	Died, '82, Nov. 15.	
R. Emm.	R. $\frac{20}{20}$ J. 1 L. blind	R. 3½ mm., L. 3; both act to light and to accommodation.	Still under observation.	
Emm.	R. $\frac{20}{25}$ J. 6 L. $\frac{20}{30}$ J. 2	R. 3 mm., L. 2¾, regular; act fairly well to light and well to accommodation.	Died, '82, Aug. 6.	
Emm.	R. $\frac{20}{30}$ J. 4 L. $\frac{20}{30}$ J. 6	R. 3 mm., L. 3½; both act to light, but right sluggish; both act to accommodation.	Died, '83, June 5.	
R. – 15 D. L. – 18 D.	R. $\frac{20}{0}$ J. 1 L. $\frac{20}{0}$ J. 1	R. 5 mm.; L. 4½ mm.; scarcely any action to light; act to accommodation.	Died, '82, Feb. 18.	Microscopical examination of optic nerves.
Emm.	R. $\frac{20}{30}$ J. 1 L. $\frac{20}{20}$ J. [illegible]	Both 2¾ mm., regular; act to light and to accommodation.	Died, '83, Oct. 17.	Microscopical examination of optic nerves, &c. Plate I. Figs. 2 and 3. Pachymeningitis of cerebral dura mater.

f the main vessels, these parts presented perfectly normal characters.

TABLE I.—SHOWING THE CONDITION OF THE EYES

No.	Initials of Patient.	Sex.	Age.	Stage of Disease.	Dates of Examinations.	Condition of Fundus, &c.
13	C. B. *contd.*					fundus, the redness invading the physi logical cups; edges decidedly blurre though not equally so everywhere; out edge of right pretty distinct for a sho distance.
				1st	'82, Mar. 1	Much the same condition.
				„	„ May 3	Discs decidedly red, but less so than on form examinations—left the redder of the tw slight haziness of margins.
				2nd	„ Sept. 1	Much the same condition, but redness som what less marked.
				„	'83, Jan. 25	Right disc decidedly paler, but upper zo still of a distinct pink hue; edges ve slightly blurred. Left disc still abnorm ally red; edges slightly blurred.
				„	„ April 26	Right disc pale—not pink; edges define Left disc full pink, edges defined.
				3rd	„ Aug. 22	Right disc greenish-grey throughout—no pi colour—distinctly atrophic in fact; ves not diminished in size. Left disc, pale p
14	W. D.	M.	49	2nd	'81, Dec. 17	Discs normal. Choroidal atrophy around ea with patches of black pigment; a sma recent hæmorrhage on outer margin of left round right macula minute spots of ch roidal atrophy. (Both lenses rather hazy
15	W. P.	M.	33	2nd	'81, Dec. 17	Both discs perfectly normal.
				„	'82, Mar. 8	„ „ „
16	W. K.	M.	28	2nd	'81, Dec. 21	Discs normal. Choroidal atrophy around each and one or two spots of this at periphery o left fundus.
				„	'82, Mar. 11	Same condition.
17	J. L.	M.	46	2nd	'81, Dec. 11	Discs normal. A pearly patch of op fibres on inner side of right.
18	W. A.	M.	37	1st	'81, Dec. 25	Both discs perfectly normal.
				„	'82, Mar. 11	Both discs decidedly white, greyish white inner edge of left alone somewhat pink.
				2nd	„ May 31	Right disc very pale, but slight tinge of pin on upper and inner side; left, lower an temporal portions very white, remainde pink.
				„	„ Aug. 28	Same condition.
19	P. M.	M.	42	2nd	'81, Dec. 26	Both discs perfectly normal (full red).
				„	'82, Mar. 8	Same condition.
				„	„ Dec. 28	„ „
				„	'83, Sept. 12	„ „
				3rd	'84, Feb. 20	„ „
20	W. Y.	M.	42	2nd	'81, Dec. 26	Both discs perfectly normal.
				„	'82, Mar. 8	Same condition.
				„	'83, Jan. 8	„ „
21	J. P.	M.	36	„	'81, Dec. 26	Both discs decidedly red—abnormally outer edge of left a little indistinct.
				„	'82, Mar. 11	Discs normal.

TY-SIX CASES OF GENERAL PARALYSIS OF THE INSANE (*continued*).

Refraction of Eyes.	Acuteness of Vision.	Condition of Pupils.	Termination of Case.	Remarks.
	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 2½ mm., right acts very slightly to light, left acts freely; both act well to accommodation.		
Emm.	R. $\frac{20}{70}$ J. 16 L. $\frac{20}{40}$ J. 12	Both 3½ mm., right very slightly elongated vertically, very sluggish to light, but act slightly; act slightly to accommodation.	Died, '82, Mar. 8.	
Emm.	R. $\frac{20}{30}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 3 mm., slightly elongated upwards and inwards; very sluggish action to light; act well to accommodation.	Died, '82, June 11.	
Emm.	R. $\frac{20}{30}$ J. 1 L. $\frac{20}{30}$ J. 1	R. 3½ mm., L. 3; R. acts well to light; L. sluggish; both act well to accommodation.	Died, '82, Nov 1.	
Emm.	R. $\frac{12}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 2½ mm., regular; act to light and to accommodation.	Removed from asylum, '82, Nov. 2.	
Emm.	R. $\frac{20}{40}$ J. 1 L. $\frac{20}{30}$ J. 1 R. $\frac{20}{30}$ J. 1 L. $\frac{15}{20}$ J. 1 R. $\frac{14}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 3 mm., R. acts well to light, L. sluggish; both act to accommodation.	Removed from asylum, '82, Aug. 23.	The variations in the acuteness of vision, as tested by types at different periods, was doubtless due to the mental inattention that existed during the early stage of excitement.
Emm.	R. $\frac{20}{20}$ J. 6 L. $\frac{20}{20}$ J. 6	Both 4 mm., regular; act well to light and to accommodation.	Still under observation.	
Emm.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 2 mm., regular; no action to light, but act well to accommodation.	Died, '83, Dec. 3.	
Slightly myopic.	R. $\frac{20}{30}$ J. 1 L. $\frac{20}{30}$ J. 1	Both 2 mm., regular; no action to light, act well to accommodation.	Died, '82, Sept. 11.	

TABLE I.—Showing the Condition of the Eye

No.	Initials of Patient.	Sex.	Age.	Stage of Disease.	Dates of Examinations.	Condition of Fundus, &c.
22	J. R.	M.	50	2nd	'81, Dec. 26	Discs normal (full red).
„				„	'82, Mar. 11	Same condition.
„				„	'83, Jan. 5	Right discs normal (pink); left, inner edg little blurred.
23	E. J.	F.	35	3rd	'82, Jan. 2	Both discs somewhat white, but probab normal. (A slight opacity in left lens.)
24	W. R.	M.	43	3rd	'82, Jan. 6	Discs normal.
25	P. T.	M.	49	2nd	'82, Jan. 6	Discs normal (pink).
				„	„ April 26	„ „
				„	'83, Jan. 5	„ „
26	E. C.	F.	37	1st	'82, April 12	Discs normal.
				„	'83, Oct. 28	„ „
27	J. M.	M.	42	2nd	'82, April 26	Discs normal.
28	S. B.	F.	40	1st	'81, Feb. 12	Discs normal.
				2nd	'82, April 7	Both discs distinctly atrophic; greyish-whit punched out appearance of edges; vesse both arteries and veins very small, pa ticularly the former.
				„	„ Aug. 2	Discs greyish-white; appear flat; no distin cupping; vessels as before.
				3rd	'83, Jan. 25	Both discs of a staring greyish-white; edg sharply defined; vessels as before.
29	E. M.	M.	39	2nd	'82, April 29	Discs normal (pink).
30	T. W. C.	M.	35	2nd	'82, May 31	Discs normal (right, pink; left, red).
				„	„ Dec. 28	„ „ „ „
31	J. H.	M.	38	2nd	'82, May 31	Both discs normal (pink).
32	J. H.	M.	43	2nd	'82, June 11	Both discs normal (pink). Edges of left somewhat jagged, but not obscured.
				„	'84, Feb. 20	Same condition.
33	M. K.	F.	45	2nd	'82, June 14	Both discs pale; nasal sides pale pink, tem poral sides whitish, and present a stipple appearance; choroidal disturbance on oute side of each (progressive). Some want definition at outer margin of right disc.
				„	'82 Nov. 4	Discs appear normal (pink); edges define (Cups shelve off gradually on tempor sides; lamina cribrosa well seen in each.)
34	T. W.	M.	47	2nd	'82, June 14	Both discs very red throughout—hyperæmic the redness is not simply due to diffuse capillary tint, but there appears an actu development of minute vessels; margins

TY-SIX CASES OF GENERAL PARALYSIS OF THE INSANE (*continued*).

Refraction of Eyes.	Acuteness of Vision.	Condition of Pupils.	Termination of Case.	Remarks.
Emm.	R. $\frac{20}{70}$ J. 8 L. $\frac{20}{20}$ J. 8	R. 2½ mm.; L. 3 mm.; right, no action to light; left, very sluggish; both act to accommodation.	Died, '83, Oct. 15.	
..	..	R. 2 mm., L. 3½ mm.; scarcely any action to light, but act to accommodation.	Died, '82, Jan. 26.	Microscopical examination of optic nerves. Plate I. Fig. 1.
..	..	Both 3 mm.; act to light and to accommodation, but sluggish.	Died, '82, Mar. 11.	Indirect examination only. Large ulcers on corneæ. Microscopical examination of optic nerves.
Emm.	R. $\frac{20}{30}$ J. 1 L. $\frac{20}{50}$ J. 1	Both 2½ mm., regular; act fairly well to light, and well to accommodation.	Died, '83, July 8.	
Emm.	R. $\frac{20}{30}$ J. 1 L. $\frac{20}{30}$ J. 1	Both 3 mm.; scarcely any action to light.	Still under observation.	
R. Emm. L., simple myopic stigmatism.	R. $\frac{20}{30}$ J. 1 L. $\frac{20}{100}$ J. 1	Both 3 mm.; right acts well to light; left, no action; both act to accommodation.	Died, '82, April 26.	Accidentally suffocated by food getting into larynx. Microscopical examination of optic nerves.
..	.. Could not count fingers accurately.	.. .. Both 3 mm., regular; very sluggish to light, but act to accommodation.	Died, '83, Mar. 14.	Microscopical examination of optic nerves. Plate II. Figs. 4 and 5.
Emm.	..	R. 2½ mm., L. 3, regular; scarcely any action to light, but act to accommodation.	Died, '82, Aug. 2.	
..	..	R. 3 mm., L. 2½; right slightly irregular; both act fairly well to light; very active to accommodation.	Died, '83, Sept. 9.	
..	..	R. 4 mm., L. 2½, regular; no action to light; act to accommodation.	Died, '83, June 23.	
R. + 2 D. L. + 2 D.	..	Both 3½ mm., regular; act well to light and to accommodation.	Still under observation.	
R + 3 D L + 3 D	R. $\frac{20}{30}$ J. 1 L. $\frac{20}{50}$ J. 1	Both 1¾ mm.; no action to light; act well to accommodation.	Died, '82, Nov. 11.	Microscopical examination of optic nerves.
+ 1 D L + 1 D	R. $\frac{20}{30}$ J. 4 L. $\frac{20}{50}$ J. 4	Rt. 3 mm.; left 3½; regular; act well to light and to accommodation.	Removed from Asylum, '83, Aug. 27.	

TABLE I.—Showing the Condition of the Eye

No.	Initials of Patient.	Sex.	Age.	Stage of Disease.	Dates of Examinations.	Condition of Fundus, &c.
34	T. W. *contd.*					little misty, especially at upper part; fine white line crosses inner side of le disc; ? exudation. Pigmentary disturban on outer side of each.
				2nd	'83, Jan. 8	Both discs red, but probably less so than whe last examined; upper and inner edge right disc is slightly ill-defined, but elsewhere edges are quite clear.
35	J. G.	M.	36	1st	'82, Aug. 24	Both discs normal (pink); edges well-define
				2nd	'83, Jan. 8	Both discs perfectly normal.
36	H. K.	F.	40	2nd	'83, April 25	Right disc palish; margins somewhat indistinct; vessels normal. Left disc fla whitish; margins pretty distinct; vesse very small. Advanced condition of retinit pigmentosa in left eye. Floaters in le vitreous. Cortical striæ in each lens, mo marked in left.
37	G. O.	M.	59	1st	'82, Nov. 14	Right disc very red, but perhaps not abnormally so; edges defined. Left, large glaucomatous cap, surrounded by a narrow rin which is probably formed by the sclerotic.
				”	'83, Jan. 11	Same condition.
				”	'83, April 26	Same condition.
				2nd	'84, Feb. 20	Same condition, but pulsation of arterie noted in right eye. Some increase of tension in both globes.
38	J. B.	M.	36	2nd	'82, Nov. 14	Right disc pale pink; vessels rather small white tissue around vessels; edges of dis defined. Left disc very pale; vessels decidedly small; much white tissue aroun vessels. Edges defined.
39	W. R.	M.	28	1st	'82, Dec. 26	Right disc red throughout; outer margi quite clear and distinct; inner margi slightly softened, though edge can b traced; white tissue very conspicuou around some of the vessels, even the very small ones; a patch of white tissue arounc a central artery just at its bifurcation is very conspicuous. Left disc much the same condition, but tint is more pink than red some slight blurring of inner margin; white lines along vessels distinct, but not so well marked as in right. Vessels well filled in each
				2nd	'83, Sept. 12	Same condition.
				3rd	'84, Feb. 20	Right disc pink; inner margin a little indistinct (scarcely any physiological cup). Left. pink; inner margin a little indistinct. Vessels normal in each.
40	H. T.	M.	40	2nd	'82, Dec. 26.	Both discs rather red, left the more so; edge well defined; vessels normal.
41	G. McG.	M.	49	2nd	'82, Dec. 28	Both discs full red, but redness does not invad physiological cups, and edges are defined.
42	C. A. W.	M.	37	1st	'83, Jan. 10.	Both discs decidedly red throughout; edges defined; white tissue around vessels.
				”	'83, April 26	Both discs very red throughout; rednes striated; margins run into retina; a littl white tissue around vessels.
				2nd	'83, Sept. 12	Much the same; redness very well marke, and margins of discs very indistinct to direct examination.

ty-six Cases of General Paralysis of the Insane (*continued*).

Refraction of Eyes.	Acuteness of Vision.	Condition of Pupils.	Termination of Case.	Remarks.
Emm.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	R. 3 mm., L. 2¾, regular; act well to light and to accommodation.	Died, '83, July 30.	
R. Emm. L − 5 D	..	R. 4¼ mm., L. 3, regular; scarcely any action to light, but act to accommodation.	Still under observation.	
..	R. $\frac{20}{30}$ J. 12 L. blind	..	Still under observation.	
Emm.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	..	Died, '83, Feb. 24.	
Emm.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	R. 4 mm., L. 4¼, regular; act very slightly to light, but well to accommodation.	Still under observation.	
Emm.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	..	Died, '83, April 12.	Microscopical examination of optic nerves.
Emm.	..	..	Died, '83, May 23.	
..	..	Both 3 mm.; act to light and to accommodation.	Died, '83, Nov. 16.	Died of pneumonia. Microscopical examination of optic nerves, etc. Plate II. Fig. 6.

TABLE I.—Showing the Condition of the Ey

No.	Initials of Patient.	Sex.	Age.	Stage of Disease.	Dates of Examinations.	Condition of Fundus, &c.
43	A. K.	M.	54	2nd	'83, Jan. 11	Both discs pale, right decidedly so; ves both arteries and veins distinctly small.
44	P. W.	M.	38	2nd	'83, Jan. 13	Right disc of a well-marked grey col throughout, distinctly atrophic; vessels d tinctly diminished in size, especially t arteries; a little choroidal disturbance upper and outer part. Left, of a pale g tint throughout; vessels slightly diminish in size, especially the arteries.
				„	'83, April 26	Both discs very white—have a punched c appearance, and appear somewhat ex vated. Vessels diminished in size.
45	J. W.	M.	40	2nd	'83, Jan. 28	Right disc very pale; vessels distinctly dim ished in size. Left disc (seen only by i direct examination) appeared normal (pin Both lenses very hazy, and in right a d tinct peripheral flocculent opacity on ou side.
46	R. B.	M.	56	2nd	'83, April 28	Both discs normal (pink); vessels normal.
47	W. W.	M.	35	1st	'83, April 28	Both discs normal (pale pink); vessels n mal.
				2nd	'84, Feb. 20	Same condition.
48	S. Y.	M.	29	2nd	'83, April 28	Both discs very pale, with a very slight tin of pink; R. the paler of the two; edg very clear and defined. Vessels perfect normal.
49	S. B.	M.	35	2nd	'83, April 28	Both discs normal (pink); vessels normal.
50	W. C.	M.	57?	2nd	'83, June 23	Both discs perfectly normal (pink).
51	R. N.	M.	50	1st	'83, June 23	Both discs apparently normal, but edges trifle hazy, especially in left; colour discs, pink. (?) Arteries somewhat smal Floaters in each vitreous.
				„	'84, Feb. 20	Both discs red; margins a little misty feathery. Arteries distinctly small.
52	J. B.	M.	40	1st	'83, June 23	Both discs normal (pale pink); vessels no mal. A small spot of pigment on posterio surface of L. lens.
				„	'84, Feb. 20	Same condition.
53	A. B.	M.	32	1st	'83, June 23	Right disc decidedly grey—greenish grey edges very clear; slight hollowing; vesse normal. L. same condition, but a litt less marked.
				„	„ Nov. 23	Much the same condition, but, if anything more marked; L. is, perhaps, now a litt the paler of the two.
				2nd	'84, Feb. 20	Both discs greenish grey throughout. Art ries distinctly small.
54	P. McG.	M.	52	3rd	'83, June 23	Both discs normal (pale pink). Lenses muddy striæ at lower and inner part periphery of each.

TY-SIX CASES OF GENERAL PARALYSIS OF THE INSANE (*continued*).

Refraction of Eyes.	Acuteness of Vision.	Condition of Pupils.	Termination of Case.	Remarks.
..	R. $\frac{20}{70}$ J. 16 L. $\frac{20}{50}$ J. 16	..	Died, '83, June 3.	
Emm.	R. $\frac{20}{200}$ J. 19 L. $\frac{20}{70}$ J. 8	Both 1¾ mm., regular; no action to light, but act to accommodation.	Died, '83, May 5.	Died of pneumonia. Had clinical signs of locomotor ataxy, and after death grey degeneration of posterior columns of cord was found throughout greater portion of length. Optic nerves distinctly wasted to naked eye examination.
..	..	..	Died, '83, July 28.	Had clinical signs of lateral sclerosis.
Emm.	..	R. 3 mm., L. 2½, regular; no action to light, act fairly well to accommodation.	Died, '83, July 29.	
Very slight myopic stigmatism.	R. $\frac{20}{30}$ J. 1 L. $\frac{20}{30}$ J. 1	R. 3 mm., L. 4; R. acts very slightly to light, L. not at all; both act fairly well to accommodation.	Still under observation.	
Emm.	..	..	Died, '83, Oct. 8.	
Emm.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	R. 3 mm., L. 2¾, regular; do not act to light, but act to accommodation.	Died, '84, Jan. 8.	
Emm.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 2¾ mm., regular; act to light, but sluggish; act well to accommodation.	Removed from Asylum, '83, July 23.	
Emm.	R. $\frac{20}{30}$ J. 14 L. $\frac{20}{30}$ J. 14	Both 2¾ mm., regular; sluggish action to light; act well to accommodation.	Still under observation.	
Emm.	R. $\frac{20}{20}$ J. 1 L. $\frac{20}{20}$ J. 1	Both 1¾ mm., regular; no action to light, but act to accommodation.	Still under observation.	
Emm.	R. $\frac{20}{30}$ J. 6 L. $\frac{20}{30}$ J. 6	Both 3 mm., regular; no action to light, and very little to accommodation.	Still under observation.	Partial paralysis of 3rd nerve in right eye, indicated by external strabismus and slight ptosis.
..	..	..	Died, '83, Aug. 8.	

TABLE I.—Showing the Condition of the Eye

No.	Initials of Patient.	Sex.	Age.	Stage of Disease.	Dates of Examinations.	Condition of Fundus, &c.
55	J. T.	M.	42	2nd	'83, June 23	Both discs normal (pink). R., well-mark appearance of opaque fibres surrounding t disc, the margins of which, however, a defined, the opacity not invading the su face of the disc itself. L., considerab pigmentary disturbance at inner and out margins.
				"	'84, Feb. 20	Same condition.
56	T. H.	M.	42	2nd	'83, June 23	Both discs normal (pink).
				"	'84, Feb. 20	Same condition.
57	H. O.	F.	44	2nd	'83, Aug. 22	Both discs normal (pale pink); arteries rath small; choroidal disturbance around righ floaters in both vitreous bodies; conic cornea in left eye.
58	J. N.	M.	43	2nd	'83, Sept. 4	Both discs pale pink; edges defined; wel marked sclerotic rim, forming a sma crescent on outer side; vessels a little sm especially on left disc.
59	H. E.	M.	42	2nd	'83, Oct. 27	Both discs perfectly atrophic; surface fla edges blurred; veins very slightly dimi ished in size, arteries considerably so.
				"	'84, Feb. 20	Same condition; veins much diminished left disc. Numerous small pigment spo in retina.
60	R. B.	M.	36	2nd	'83, Nov. 23.	Both discs full red; inner edges slight softened. R., well-marked crescent at out and lower parts, and small one on inne L., small crescent at outer and lower part
61	M. A.	F.	45	2nd	'83, Dec. 8	Both discs normal (full pink).
				"	'84, Feb. 23	Same condition.
62	E. T.	F.	25	1st	'84, Jan. 19	R. disc, margins very indistinct; vesse diminished, the arteries being very fine retinitis pigmentosa, the pigment exten ing quite up to disc; one or two floaters i vitreous. L. disc, and retina in same con dition, but less marked; disc red in centre the pigment extends only to the middl zone of retina.
63	M. O.	F.	38	1st	'84, Jan. 19	Both discs normal (red); vessels normal. slight granular appearance at R. macul and at L., pale buff patches finely sprinkle with black.
				"	" Feb. 23	Same condition.
64	M. R.	M.	32	1st	'84, Jan. 20	Both discs pale pink; R. the paler of the tw A small, irregular-shaped crescent aroun each, especially at outer part. Middle zon of each fundus covered with well-marke circular, pigmented, white patches of ch roidal inflammation, ? syphilitic, mos marked in L.; floaters in L. vitreous.
65	W. W.	M.	52	2nd	'84, Jan. 20	Both discs normal (red).
66	S. F.	F.	23	3rd	'84, Feb. 2	Both discs normal (pink).

y-six Cases of General Paralysis of the Insane (*continued*).

efraction f Eyes.	Acuteness of Vision.	Condition of Pupils.	Termination of Case.	Remarks.
−1 D −1 D	..	Both 3 mm., regular; act to light and to accommodation.	..	
			Still under observation.	
Emm.	..	..	Still under observation.	
+ 2 D. + 3 D.	..	..	Died, '84, Feb. 22.	
..	..	Both 3 mm., regular; act to light, but sluggish; act well to accommodation.	Died, '83. Nov. 28.	
Emm.	Blind	Both 4 mm.; no action to light; act slightly to accommodation.	Still under observation.	Has signs of sclerosis of lateral columns of cord.
ightly + ach eye.	..	..	Died, '83, Dec. 7.	A case which ran a very rapid course.
Emm.	..	R. 2½ mm., L. 2. R. acts very slightly to light; L. not at all. Both act to accommodation.	Still under observation.	
Emm.	..	R. 3 mm., L. 3½. Both act to light, but sluggish; act to accommodation.	Still under observation.	R., well-marked nebula about centre of cornea; on anterior surface of lens, a cluster of small brownish spots. L., two faint nebulæ on cornea, one or two old adhesion bands, and an appearance as of new growth around margin of dilated pupil.
R. simple astigmatism. Emm.	R. $\frac{20}{50}$ J. 4 L. $\frac{20}{30}$ J. 1	Both 3 mm., regular; act to light and to accommodation.	Still under observation.	
− 3 D. − 3 D.	..	..	Still under observation.	
..	..	R. 4 mm., irregular; L. 3, act slightly to light.	Still under observation.	
− 5 D. Emm.	..	R. 5 mm., L. 4; both act to light.	Still under observation.	Has signs of lateral sclerosis.

TABLE II.—Showing the Condition of the Optic Discs, &c., in Twelve cases of Mania, in which Slight (or Doubtful) Abnormalities were Detected.

No.	Sex.	Age.	Form of Mania.	Condition of Fundus, &c.
1	F.	38	Acute (recent)	Both discs pink, edges perhaps a little hazy; a spot of pigment on outer edge of right.
2	F.	53	„ „	Both discs very red, about the same colour as the fundus; edges a little blurred.
3	F.	42	„ „	Both discs red and hazy. ? Vessels a little small. Well-marked syphilitic choroiditis in each eye, all over fundus; gross changes; large white patches mottled with black.
4	M.	42	„ „	Both discs decidedly red, right the redder of the two, and the margin of this, especially at upper part, is not very clearly defined (probably on account of similarity of colour between it and fundus).
5	F.	23	Puerperal	Both discs perfectly normal; arteries rather small; *veins abnormally full* (a second examination made after an interval of 11 months, when patient was convalescent, showed the veins to be full, but not abnormally so; condition otherwise unchanged).
6	F.	29	„	*1st examination.*—Both discs full red; edges well-defined; striation of small vessels distinct. *2nd examination* (12 days after 1st).—Much the same condition; substance of disc has a slightly softened appearance.
7	F.	30	„	*Examination 24 hours before death.*—Both discs red, but redness does not invade central cups; colour of left disc almost that of surrounding fundus, but edge can be traced, though it is somewhat indistinct.
8	F.	22	„	Both discs red throughout; upper and inner portion of left disc slightly misty. (Note.—*Patient had mitral stenosis.*)
9	F.	30	„	Both discs decidedly red, but edges are perfectly defined. (*Indirect examination only; patient very excited.*)
10	F.	41	Chronic	R. disc greyish-white; vessels decidedly small; enormous staphyloma around disc developed chiefly to outer side, extending by a series of spots of partial atrophy, as far as macula; small central opacity in posterior capsule of lens; numerous floating bodies, large and small in vitreous, which fall rapidly; −12 D. L. disc has a faint tinge of pink; vessels larger than in right; a small spot of atrophy in vicinity of macula; small floaters in vitreous; −7 D.
11	F.	65	„	Both discs normal (pink); vessels small, especially arteries (both lenses hazy, and marked cortical striæ in each).
12	F.	34	Delusional (monomania.)	Discs somewhat red, and edges a little indistinct.

TABLE III.—SHOWING THE CONDITION OF THE OPTIC DISCS, &C., IN NINE CASES OF MELANCHOLIA, IN WHICH SLIGHT (OR DOUBTFUL) ABNORMALITIES WERE DETECTED.

No.	Sex.	Age.	Form of Melancholia.	Condition of Fundus, &c.
1	F.	46	Active (recent)	Discs normal (pale pink), *Arteries* full—as large as veins or nearly so. Pigmentary deposit on outer side of each disc.
2	F.	27	,, ,,	Discs decidedly red, but probably normal.
3	F.	55	Passive (recent)	Right disc palish, flat; margins a little indistinct; arteries small; left disc whitish; margins a little blurred; appears flat. Vessels, both arteries, and veins, decidedly small. [History of chronic alcoholism.]
4	F.	30	,, ,,	Both discs normal (pale-pink); vessels, both arteries and veins, rather small.
5	M.	46	,, ,,	Both discs pale, with a shade of pink. (Three examinations made at intervals of 3 or 4 months; same condition each time, except that on first examination, the arteries were thought to be a little small; they were however normal on subsequent examinations) On anterior surface of capsule of right lens, very small brown pigment spots. [This case has some of the symptoms of General Paralysis, and that disease cannot at present be confidently excluded.]
6	F.	42	,, ,,	Both discs pale pink; edges slightly misty; vessels a little small; several recent hæmorrhages, small, and of moderate size, in both retinæ, not far from disc in right, especially about macula; one or two indistinct white patches in right retina, but there are several of these well marked in left. [Patient has chronic Bright's disease.]
7	F.	32	,, ,,	Both discs very pale; distinctly anæmic. [Patient masturbates excessively.]
8	F.	53	Passive (chronic)	Inner side of each disc, especially left, has a peculiar variegated or speckled appearance, due to the presence of five white lines or streaks; they otherwise appear normal; white lines very marked along some of the vessels. A pigmentary deposit at upper part of left fundus. (Two examinations made at an interval of 11 months—condition each time practically the same.)
9	F.	35	,, ,,	Both discs normal; a small horizontally-placed recent hæmorrhage on inner side of left disc.

TABLE IV.—SHOWING THE CONDITION OF THE OPTIC DISCS IN TWO CASES OF MENTAL STUPOR DURING THE ATTACK AND AFTER CONVALESCENCE.

No.	Sex.	Age.	——
1	F.	29	1*st examination—during condition of stupor.* Both discs perfectly normal (pink). 2*nd examination—during lucid interval.* R. disc red, L. more pink than red; edges quite clear and defined; vessels normal. (Hypermetropic astigmatism in each eye.)
2	F.	40	1*st examination—during condition of stupor.* Both discs pale; edges well defined. 2*nd examination—state of mental hebetude during convalescence.* Both discs pink, inclining to pale. 3*rd examination—after convalescence.* Both discs still palish, as in last note.

TABLE V.—SHOWING THE CONDITION OF THE OPTIC DISCS, &C., IN FIFTEEN CASES OF DEMENTIA IN WHICH ABNORMALITIES WERE DETECTED, THE MAJORITY BEING SLIGHT OR DOUBTFUL.

No.	Sex.	Age.	Form of Dementia.	Condition of Fundus, &c.
1	F.	64	Secondary to Mania.	Both discs colour of fundus, but edges distinct, except so far as indistinctness results from similarity of colour between disc and choroid; vessels a little small. (Patient has granular kidneys.)
2	F.	39	”	Discs normal (pale); arteries rather small.
3	F.	27	”	R. disc very red throughout; edges a little ill-defined; a band of white tissue stretching across central part of disc in a horizontal direction. L. disc, red; very slight want of definition about edges; no white tissue; vessels in each well filled.
4	F.	22	”	Both discs rosy red; edges of left perhaps slightly hazy to direct examination; veins large and dark; patch of pigment on inner side of right disc, and a cluster of pigment patches surrounded by an irregular greyish-white patch in vicinity of R. macula, over one of which patches a small artery can be traced by direct examination. (−7 D. each eye.)
5	F.	32	”	Both discs very red; margins slightly misty (two examinations at intervals of 7 months; same condition each time).

TABLE V.—Showing the Condition of the Optic Discs, &c., in Fifteen cases of Dementia in which Abnormalities were Detected, the Majority being Slight or Doubtful (*continued*).

No.	Sex.	Age.	Form of Dementia.	Condition of Fundus, &c.
6	F.	31	"	Both discs very red, and edges appear somewhat ill-defined, especially inner edge of left; vessels normal.
7	F.	53	"	Both discs pearly white; in both, arteries exceedingly fine, threadlike, and few in number; veins also rather small. R., white spots at macula of fair size, also a few to outer side of macula; below and to outer side of disc is a punched-out spot of choroidal atrophy, with fine retinal vessel passing over it; smaller and fainter spots in the vicinity of this. L., minute white spots around macula. (Patient has chronic Bright's disease.)
8	M.	50	"	Both discs red throughout; the slightest possible softening of edges. (Two examinations at interval of 2 months; same condition each time.)
9	F.	28	Primary (recent and chronic).	Both discs normal; tint inclining to pale; arteries rather small.
10	F.	30	" "	Both discs very red; on inner side the surface of each disc has a slight velvety appearance (most marked in left); edges perfectly clear and defined.
11	F.	28	" "	Both discs very red; white lines very distinct along some of the vessels.
12	F.	49	Organic.	R. disc greyish, and slightly swollen at the lower part; vessels rather small; three large recent hæmorrhages on inner side of disc; a large, irregular-shaped patch of choroidal atrophy, about size of disc, on outer side of this. L. disc, swollen; inner edge cannot be seen, outer very faint—quite woolly; veins large, white lines around; a large recent hæmorrhage on inner side of disc. [Patient had chronic Bright's disease.]
13	M.	47	"	R. disc very red; some pigmentary disturbance around chiefly on outer side; Left disc, similar conditions but less marked. [Patient had R. hemiplegia and aphasia.]
14	F.	67	Senile	Both discs very red; slight crescent at outer side of each most marked in R.; lenses; hazy, with cortical striæ. (R. eye, −5 D.)
15	M.	61	"	Both discs pale pink; vessels a little small, especially the arteries; discs have a hollowed appearance; lenses; slight cortical opacities.

TABLE VI.—SHOWING THE CONDITION OF THE OPTIC DISCS, &C., IN SEVEN CASES OF INSANITY ASSOCIATED WITH EPILEPSY, IN WHICH SLIGHT (OR DOUBTFUL) ABNORMALITIES WERE DETECTED.

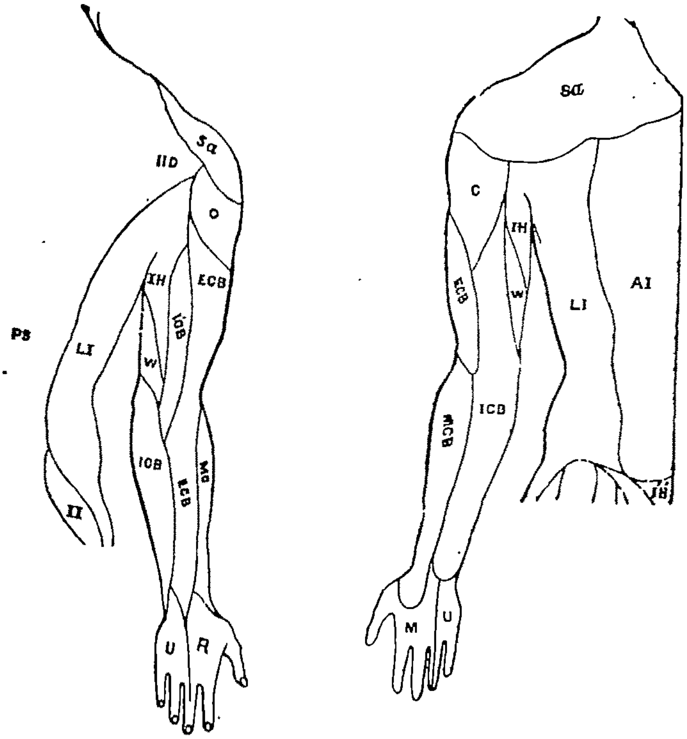
No.	Sex.	Age.	Form of Mental Disorder.	Condition of Fundus, &c.
1	F.	28	Imbecility	Right disc distinctly hyperæmic in centre. (Left disc could not be examined owing to patient's restlessness.)
2	F.	17	"	Both discs normal (rosy-red); edges well defined; arteries a little small; veins full.
3	F.	20	"	Both discs very red, the redness invading slightly the physiological cups. Right eye a few scattered yellowish-white spots a little above macula; left, the same, but running into macula, and in addition two or three spots between macula and disc.
4	F.	37	"	Both discs very red—abnormally so; margins very slightly blurred; very well marked white lines along vessels—best seen in right disc.
5	F.	12	"	Both discs full red. On inner side of right disc, a distinct white streak.
6	M.	48	Slight Dementia.	Both discs normal (pink); arteries small.
7	F.	32	Profound Dementia.	Both discs pale, the right being the paler of the two; probably normal. (Two examinations at an interval of 3 weeks—same condition each time, except that on second examination, a minute hæmorrhage was noted on an artery on right side.)

DISTRIBUTION OF ANÆSTHESIA IN CASES OF DISEASE OF THE BRANCHES AND OF THE ROOTS OF THE BRACHIAL PLEXUS.

BY JAMES ROSS, M.D., LL.D., F.R.C.P.

THE following is the account which is usually given in anatomical works of the distribution of the cutaneous nerves of the upper extremity (Figs. 1 and 2). The inner surface of the arm and forearm, as well as the adjoining portions of

FIG. 1. FIG. 2.



Figs. 1 and 2 (after Flower). Cutaneous nerves of the trunk and upper extremity.

the anterior and posterior surfaces, are supplied by the intercosto-humeral (I H), the nerve of Wrisberg (W), the internal cutaneous (I C B), and branches of the ulnar (U). All these nerves are derived from the roots of the eighth cervical, and first and second dorsal nerves. The superior portion of the shoulder is supplied by the supra-clavicular and supra-acromial branches (S A) of the cervical plexus; and the inferior part of the surfaceof the shoulder, as far as the insertion of the deltoid, is supplied by branches of the circumflex nerve (C), which is most probably derived from the fifth cervical root. The outer surface of the arm and forearm, as well as the adjoining portions of the anterior and posterior surfaces, is supplied by the internal (I′ C B) and external (E C B) cutaneous branches of the musculo-spiral nerve, and the cutaneous branches of the musculo-cutaneous nerve (M C), the musculo-spiral and musculo-cutaneous nerves being most probably derived from the fifth, sixth, and seventh cervical roots. The back of the hand is usually said to be supplied by the radial (R) and ulnar (U) nerves, the radial supplying three and a half fingers, and the ulnar one and a half. The front of the hand is supplied by the median (M) and ulnar (U) nerves, the former supplying three and a half fingers, and the latter one and a half. The skin of the ball of the thumb is also supplied by branches of the musculo-cutaneous and radial nerves. The account which Krause gives of the distribution of the digital branches of the nerves of the hands differs in important particulars from that usually given in English anatomical works; and we shall immediately see that the former corresponds much better than the latter with the results of pathological observation. "Each finger," says Krause,[1] "is supplied with two palmar and two dorsal nerves, which are derived from the principal nerve trunks to the hand, and run along the radial and ulnar borders of the front and back of the fingers. The palmar are much larger than the dorsal branches; they run along the inner side of the palmar digital arteries to reach the tips of the fingers, and are chiefly of anæsthesia caused by section of a cutaneous nerve, these experimenters found that section of one of the four nerve branches by which a digit of the dog or the cat is supplied, causes no recognisable diminution of its cutaneous sensibility, and that even section of two causes only slight general diminution of sensibility. Section of three branches, however, causes a very decided diminution of cutaneous sensibility, but complete anæsthesia is only established when all four branches are divided. Again, they found that on the fifth digit of the dog, which is supplied by branches from both the ulnar and radial nerves, partial anæsthesia only is caused by section of one or other of these nerves, but complete anæsthesia is caused by section of both nerves. In the cat, the fifth digit is supplied by branches of the ulnar nerve only, and consequently section of this nerve renders it completely anæsthetic. It is needless to enter into the details of these experiments; the general conclusion arrived at may be expressed by saying that when the four nerves of a digit are supplied from one only of the three nerves—the ulnar, the radial, and the median—which supply the skin of the paw, section of that nerve will render it completely anæsthetic; and when a digit is supplied from two nerves, both, and from three nerves, all three, must be divided before complete anæsthesia is established. This want of functional independence of the cutaneous nerves is easily explained by the free anastomosis which is known to take place, first, between branches of different nerves of considerable size, and second, between these nerves in the terminal networks described by Jacubowitsch and Beale. Arloing and Tripier have described a third method, by means of which nerve fibres from one nerve district may be distributed to another and far distant territory. After division of one of the branches of the median nerve of the dog or cat, for instance, they found that the animal gave manifest evidence of pain when the peripheral portion of the divided branch was pinched, and even division of the whole nerve did not entirely prevent these manifestations of sensibility. The peripheral portion of the divided branch was cut out and examined microscopically a few days after the section, and it was found that, although by far the greater number of the fibres were degenerated, yet a few of them had remained healthy. A portion of the proximal end of the divided nerve was also examined microscopically, and a few degenerated fibres were discovered amongst a large number of healthy ones. From these observations, the authors conclude that the few fibres which remained healthy in the peripheral portion of the nerve still retained their connection with their trophic centres, while the few which were degenerated in the proximal portion were severed from their centres. As the trophic centres of these fibres must have been situated in the spinal ganglia or in the grey matter of the cord, they must have reached the divided branch from the peripheral side of the point of section, and have been pursuing a centripetal course, and the authors justly assume that these fibres conferred recurrent sensibility upon the peripheral part of the nerve.

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[1] Krause (Carl. F. T.), 'Handbuch der menschlichen Anatomie,' 8^{e} Aufl. Hannover, 1879, s. 896. See also Henle, 'Handbuch der Nervenlehre des Menschen; Braunschweig,' 1871, s. 499.

It is not known how these fibres terminate, but Arloing and Tripier believe that they only ascend a short distance in the nerve, and it may be assumed that they are distributed to the skin and thus take an important part in supplying one district with nerve-fibres from neighbouring nerve-territories.

If we turn to pathological observation, it will be seen that in man the extent of the anæsthesia caused by division of a cutaneous nerve, either by surgical operation or injury, does not by any means correspond with the supposed anatomical distribution of the nerve.

In two cases of complete division of the ulnar nerve observed by Létiévant,[1] caused by a wound of the wrist, and in which all the muscles supplied by the nerve were completely paralysed and atrophied, the skin of the little finger and the inner half of the ring-finger, on their palmar and dorsal aspects, as well as that over the hypothenar eminence and the corresponding part on the back of the hand, was more or less anæsthetic. The least degree of anæsthesia was met with in a narrow border which formed the transition between the anæsthetic and healthy skin, and which is indicated in Figs. 4 and 5 by a light degree of shading (B). A patch of skin on the border of the hand near the hypothenar eminence, and which is indicated in Figs. 4 and 5 by the deepest shading (A), was completely anæsthetic, but in the remaining portion of skin, and which is indicated by the medium degree of shading, the sensibility was only considerably diminished.

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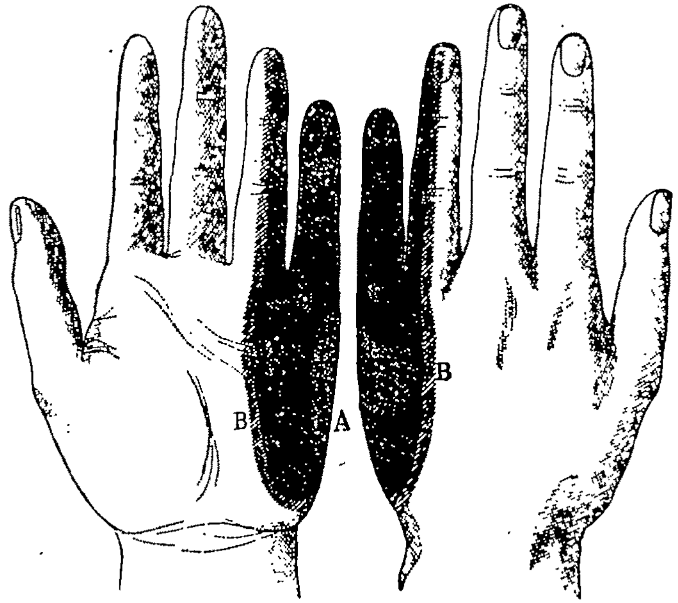
[1] Létiévant (E.), 'Traité des sections nerveuses.' Paris, 1873. (Obs. xxviii.) p. 70.

In this case the extent of the anæsthesia corresponds pretty acurately with the supposed anatomical distribution of the cutâneous branches of the divided nerve, but although the nerve was completely divided at the wrist, the anæsthesia of even the little finger, which is supposed to be wholly innervated by the ulnar nerve, was incómplete.

FIG. 4. FIG. 5.



Figs. 4 and 5 (after Létiévant). A, Deep shading, representing complete anæsthesia; B, light shading, represents slight, and C, medium shading, represents a medium degree of anæsthesia.

In another case,[1] reported by the same observer, a man aged 51 years had a wound of the wrist by a fall on a broken bottle, when he was only ten years of age. A deep cicatrix was visible above the wrist, and all the muscles of the hand supplied by the ulnar were completely paralysed and atrophied, but there was only a slight blunting of the sensibility in the region of distribution of the cutaneous branches of the injured nerve.

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[1] Létiévant (E). Op. cit., Obs. xxix., p. 73.

This case appears to show that the anæsthesia caused by complete division of one of the cutaneous nerves of the hand diminishes in intensity with the lapse of time, and we shall hereafter find abundant evidence to show that it becomes diminished in extent also, and that the healthy territory gradually encroaches upon the affected district, so that the area of the latter becomes much restricted in the course of time.

The effects of division of the median nerve on the sensibility of the hand are no less interesting and instructive than those of injury of the ulnar.

FIG. 6.

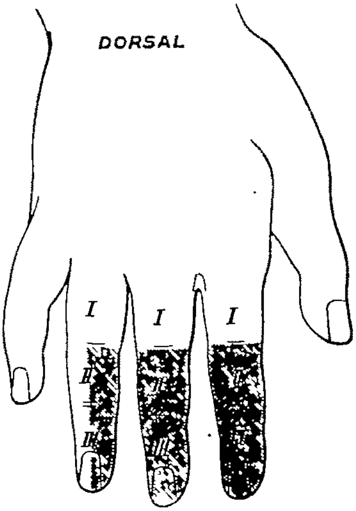

Fig. 6 (after Bernhardt), showing the distribution of the anæsthesia on the back of the fingers in a case of injury of the median nerve.

The case of a man aged 59 years, is reported by Bernhardt,[1] who sustained a fracture of the styloid process of the ulna, and severe concussion of the left forearm and hand through a fall. During the first three weeks after the injury no special treatment was adopted, and the patient was able to move all the fingers. At the end of this time the hand was placed in a plaster-of-Paris bandage which reached upwards as far as the middle of the forearm, and in which it was retained for three weeks. Four months after the injury, the patient came for the first time under the observation of Bernhardt. The wrist-joint was then moveable in all directions, but the muscles supplied by the median nerve were found paralysed and atrophied, while those supplied by the ulnar nerve were unaffected. On the palmar aspect of the hand the sensibility of the skin of the ball of the thumb and the radial half of the palm was considerably diminished, whilst that of the thumb, of the index, middle, and radial border of the ring finger was greatly diminished, the loss of sensibility becoming progressively greater from the basal to the ungual phalanges. On the dorsal aspect the sensibility of the skin of the thumb and little finger was intact over all the phalanges, but was much diminished over both borders of the middle and ungual phalanges of the index and middle fingers, and over the radial border of the corresponding phalanges of the ring finger (Fig. 6).

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[1] Bernhardt, (M.) "Neuropathologische Beobachtungen." 'Archiv für Psychiatrie und Nervenkrankheiten,' Bd. v., 1875, p. 558.

From five cases of division of the median nerve, carefully observed by Létiévant[1] he concluded that the anæsthesia is distributed over the palmar surfaces of the thumb, index and radial border of the middle finger, the thenar eminence, and the radial half of the palm, and over the dorsal surfaces of the ungual and middle phalanges of the index and middle fingers, and occasionally the ungual phalanx of the thumb. The anæsthesia is only partial except over the palmar surface of the whole of the ungual phalanx of the index finger, and part of the dorsal surface of the same phalanx. The different degrees of shading in Figs. 7 and 8 are intended to represent the varying degrees of anæsthesia, the very deep shading at A representing complete anæsthesia. It has already been remarked that the area of normal sensibility tends to encroach upon the anæsthetic area with the lapse of time; and in the following case, which came under my own observation, the area of anæsthesia resulting from division of the median was by no means so extensive on the dorsum of the fingers as in the cases reported by Bernhardt and Létiévant.

The case was that of a young man who was under the care of my colleague, Mr. Hardie, and who had received a deep wound on the front of the left forearm above the wrist, by falling on the broken edge of a glass bottle nine months before he came under my observation. The lower parts of the anterior surface of the left forearm presented a deep cicatrix which ran obliquely across the limb, and to which the tendons of the long flexors of the fingers were adherent. The thenar and hypothenar eminences were flattened, and the hand was distorted into the "claw" form, which is so characteristic of division of the ulnar and median nerves. All the intrinsic muscles of the hand failed to re-act to either a strong faradic or a galvanic current, and it may consequently be concluded that no union had taken place in either the median or ulnar nerves. The distribution of the anæsthesia is shown in Figs. 9 and 10, but I much regret that the careful notes which were taken at the time have been mislaid, so that I am unable to make any very positive assertions with regard to the diminution of the various forms of sensibility. The diagrams which were taken at the time, may be relied upon to represent accurately the extent of the anæsthetic area, and the varying degrees of the shading also represent in a general way the different degrees of anæsthesia which was present.

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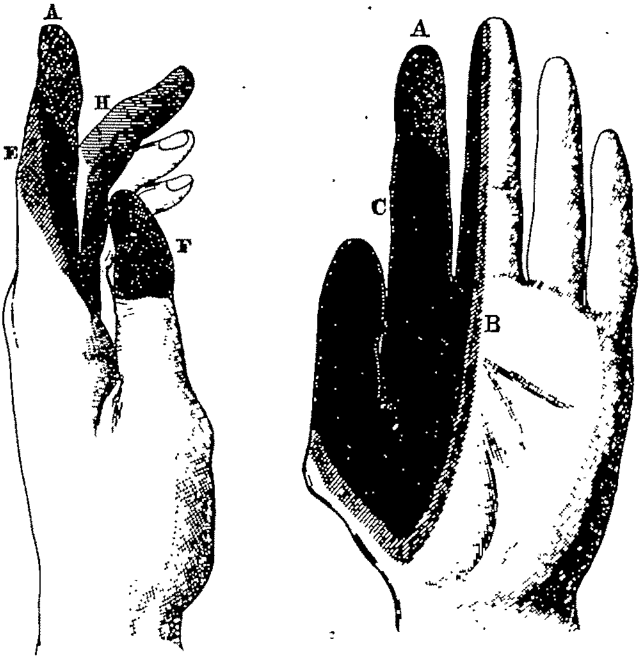
[1] Létiévant, op. cit., p. 41.

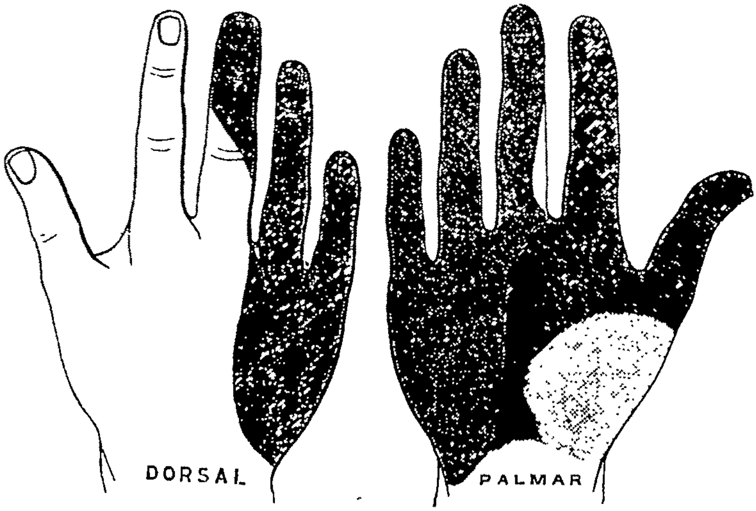
FIG. 7. FIG. 8.



Figs. 7 and 8 (after Létiévant). Radial border and dorsal aspect of the hand showing the distribution of anæsthesia in a case of division of the median nerve. A, Deep shading representing more or less complete anæsthesia; B, E, and H, light shading, and C, medium shading, representing a slight and medium degree of anæsthesia respectively.

A very important case is reported by Dr. Weir Mitchell,[1] in which a portion of the musculo-spiral and median nerves were excised, at different times, for the relief of intense brachial neuralgia in a lady. Towards the end of 1871 Dr. Sapolini, surgeon to the King of Italy, removed an inch of the musculo-spiral nerve in the upper arm. The patient was astonished at the slight diminution of tactile sensibility which had followed the operation, but the relief obtained was only of short duration, inasmuch as the pain recurred eight days afterwards. The extensor muscles remained paralysed for some time, but motor power gradually returned in them about six months after the nerve was divided, and two years after the operation, voluntary power over the extensors was scarcely impaired. The sensory disorders present thirteen months after the operation, when the lady first came under Dr. Mitchell's care, are thus described:[2] "The radial and median sensation in the arm is normal, as well as that of the musculo-cutaneous nerve. In the radial area in the

Fig. 9. Fig. 10.



Figs. 9 and 10. Dorsal and Palmar aspects of the Hand, showing the distribution of anæsthesia in a case of division of the ulnar and median nerves. The different degrees of shading show in a general manner the degrees of anæsthesia present.

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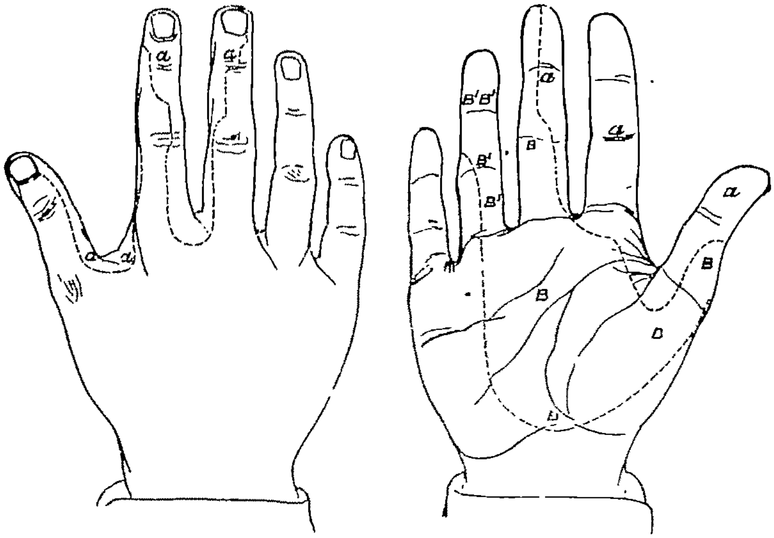
[1] Mitchell (S. Weir). "Traumatic neuralgia; Section of Median Nerve." 'The American Journal of the Medical Sciences,' vol. ii., 1874, p. 17.

[2] Mitchell. Loc. cit., p. 19.

back of the thumb and first and second fingers, touch is referred correctly, and is everywhere felt: but throughout this region every touch is felt as both touch and pain. In a less degree this is true of certain parts of the whole arm, and most notably of parts just around the scene of the operations on the musculo-spiral. The ulnar territory is in all respects healthy by compass point. The sensitiveness of the thenar eminence is not acute, but increases as we approach the index-finger. The hyperalgesia is exquisite in the face of the second finger, and the radial side of the third finger; on the face of the thumb and the palm; over the first joint of the index. In these regions touch is so much more distinctly felt as pain that the degree of health of the power of touch cannot be thoroughly studied." It is manifest from this description that not much anæsthesia had remained as the result of this operation, and the return of motor power in the extensors rendered it probable that union had taken place between the ends of the divided nerve. In 1873, fifteeen months after the first operation, three-quarters of an inch of the median was, in accordance with Dr. Mitchell's advice, excised by Dr. Brinton, and in order to prevent reunion the lower end of the nerve was doubled upon itself. The distribution of the anæsthesia to pain and touch observed at the end of two weeks is shown in Figs. 11

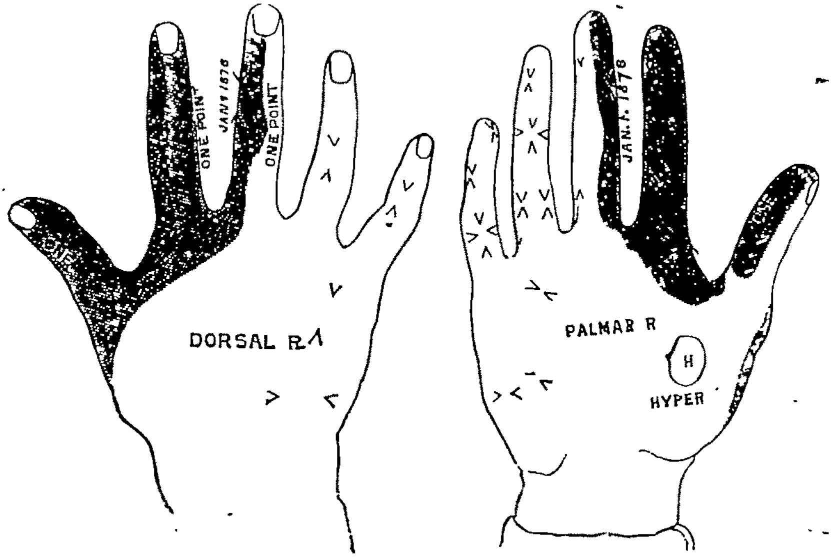
FIG. 11. FIG. 12.



Figs. 11 and 12 (after Mitchell). Dorsal and Palmar aspects of the Hand respectively; *a*, pain and touch lost; B, touch lost; B', touch slightly lessened.

and 12. When the patient was examined nine months after the operation the area of anæsthesia had become considerably diminished on the palm, but remained nearly the same on the dorsal aspect of the hand. After a time the pain returned in the territory of the musculo-spiral nerve, and Dr. Brinton, still acting under Dr. Mitchell's advice, removed nearly three inches of the nerve on a level with Dr. Sapolini's operation, which had been performed two years and seven months previously.

FIG. 13. FIG. 14.



Figs. 13 and 14 (after Mitchell). Dorsal and Palmar aspects of the Hand. The light shading indicates the area in which tactile sensibility was diminished; the deep shading the area in which it was lost. The marks (> <) indicate the appreciation of separate points. A single (V) mark indicates that the points are not felt as separate. The absence of the (V) mark indicates complete loss of tactile sensibility. H, a small area in which there is hyperalgesia of the skin.

The condition of the sensibility on the dorsal and palmar aspects of the hand, four weeks after the operation, is represented in Figs. 13 and 14 respectively. A small spot of hyperalgesia (Fig. 14, H) was observed on the thenar eminence for which it is difficult to find an adequate explanation. The precautions taken to prevent reunion of the median, taken

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[1] Mitchell (S. Weir). "Neurotomy." 'The American Journal of Medica Sciences,' vol. i., 1876, p. 322.

along with the fact that the muscles supplied by it below the point of division remained permanently paralysed, renders it extremely unlikely that its ends had become united, and no one can believe that the musculo-spiral nerve had become united four weeks after three inches of it had been excised, and consequently the terminal branches of the ulnar, and a few filaments from the internal cutaneous and musculo-cutaneous nerves had sufficed in this case to bestow sensibility upon the whole of the palmar and dorsal aspects of the hand with the exception of the comparatively small areas which are shaded in Figs. 13 and 14. The areas which are most deeply shaded in the figures, represent the regions of greatest anæsthesia, but even in these sensation was not entirely abolished, as the prick of a needle was still felt.

The disorders of sensibility caused by division of the musculo-spiral nerve are well illustrated in the case of a young soldier reported by Létiévant,[1] who had the nerve divided as the result of a wound by a poignard in the inferior and internal aspect of the forearm.

Fig. 15.

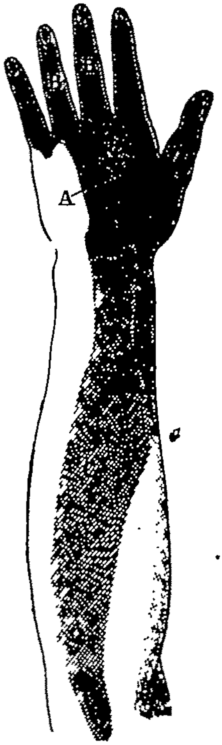

Fig. 15 (after Létiévant). Showing the distribution of the anæsthesia in a case of division of the musculo-spiral nerve in the arm. A, Deeply shaded, to show the area of greatest anæsthesia; B, shaded in a medium degree, to show the area of partial anæsthesia; C, faintly shaded, to show the area of slight anæsthesia.

The distribution of the anæsthesia on the back of the forearm and hand, two and a half years after the injury is represented in Fig. 15.

The highest degree of anæsthesia was found in a patch of skin over the back of the hand, and first phalanx of the thumb, and is represented by the deep shading (Fig. 15, A). A slighter degree of anæsthesia, represented by the medium shading in Fig. 15, B, was observed over the dorsum of the ungual phalanx of the thumb, and the back of all the fingers including the little finger, which is supposed to be innervated by the ulnar nerve. A still slighter degree of anæsthesia, represented by the light shading (Fig. 15, C), was found on the back of the forearm in the area of distribution of the external cutaneous branch of the musculo-spiral nerve.

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[1] Létiévant (E.) Op. cit. (Obs. xxxix.) p. 96.

The anæsthesia is widely distributed in this case over the back of the hand and fingers, and offers in that respect a marked contrast to the very limited area of anæsthesia in the case of resection of the median and musculo-spiral nerves reported by Dr. Weir Mitchell (Figs. 13 and 14). Another case of resection of a portion of the radial nerve is reported by Dr. Mitchell[1] in which the area of anæsthesia was of equally limited extent with his first case, although the localisation of the affected skin differed considerably in the two cases.

FIG. 16.

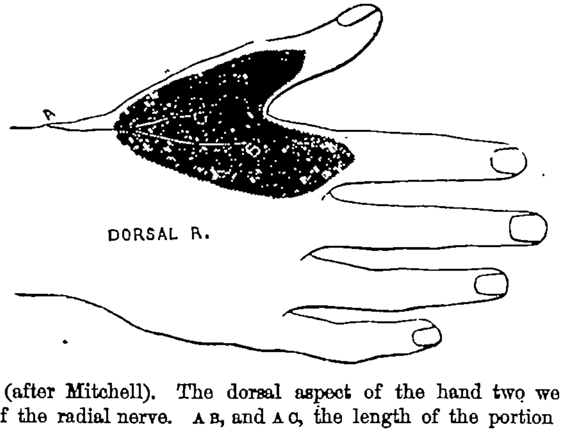

Fig. 16 (after Mitchell). The dorsal aspect of the hand two weeks after resection of the radial nerve. A B, and A C, the length of the portion removed. The light shading represents the area in which tactile sensibility was diminished; the deep shading the area in which it was lost.

In this second case, two inches of the radial nerve was excised by Dr. Hodge in the back of the forearm, for the relief of long continued and persistent pain in the back of the hand. In about six weeks the pain gradually returned, and tactile sensation, at first lost, also gradually reappeared. It was evident that the nerve had become reunited, and about ten months after the first operation three inches of the nerve were removed, and the end of the distal extremity was folded so as to prevent reunion. The distribution of the anæsthesia two weeks after the second operation is represented in Fig. 16.

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[1] Mitchell (S. Weir). Loc. cit., p. 328.

When the lesion of the musculo-spiral nerve is situated in the axilla, as in crutch paralysis, a certain degree of anæsthesia may be discovered in the anterior aspect of the forearm in the region of distribution of the superior branches of the external cutaneous nerve, and the internal cutaneous branch of the musculo-spiral, but loss of sensibility is never a marked feature of such cases. Division of the musculo-cutaneous nerve gives rise to partial anæsthesia along the radial border of the forearm, but excision or division of the circumflex nerve does not give rise to any manifest disorders of sensibility.

Sensory disorders in disease of the roots of the brachial Plexus.—Our data for determining the respective connections of the sensory nerves of the upper extremity with the cervical portion of the spinal cord are as yet very scanty. In the following case of *pachymeningitis cervicalis* at present under my care in the Barnes Convalescent Hospital, Cheadle, and in which the lesion is situated, as we shall find reason to believe, at the junction of the cervical and dorsal regions of the cord, the distribution of the anæsthesia in the upper extremities is very instructive.

Edward D——, aged 30 years, was admitted into the Manchester Royal Infirmary on October 16th, 1882, under the care of Dr. Ross.

The patient has always enjoyed good health; and, with the exception of an abscess of the testicle from which he suffered two years ago, and which troubled him for about five weeks, he has not had any illness worth mentioning up to the date of the present attack. The patient has led an unsteady life; he has many times slept, whilst in a state of intoxication, on the pavement, and has frequently gone to work during the day without changing his clothes, although he had been drenched on the previous night. His occupation is that of a dyer, but no evidence of chronic metallic poisoning can be discovered. He has not had syphilis, and no hereditary tendency to any particular disease can be found from his family history. About five months ago he began to suffer for the first time from pain between the shoulders, and at the back of the neck, which was much increased by coughing, and by movement. The pain gradually increased in intensity until it became almost unbearable; it was "stabbing" in character, and so much worse at night, and in the early morning, that he had to pass many sleepless nights. The patient had the full use of his limbs and did not feel pain or any abnormal sensation in them until about five weeks ago. At that time he felt "prickling" sensations in his feet; these gradually travelled upwards until at the end of eight or ten days, they were felt over the whole of the lower extremities and body, up to the level of the first ribs, as well as down the inner borders of the arms, forearms, and the inner halves of the hands. A feeling of numbness soon followed the prickling sensations; this feeling also began in the feet and travelled upwards until, at the end of another eight or ten days, all the parts in which the prickling sensations were previously felt had become numb and insensible to pinching. The numb feeling was accompanied by a gradual loss of power of the lower extremities, so that the patient was soon unable to walk, and he likewise observed that his grasp was feeble. He was also troubled with a severe pain in the form of a belt passing round the abdomen, and with jerkings of the lower extremities, the latter being most troublesome at night.

Present Condition.—The patient lies on his back in bed in a helpless condition. The lower extremities are completely paralysed, passive movements of them provoke muscular tension, the cutaneous reflexes are increased, the patellar-tendon reactions are exaggerated, ankle clonus can be readily elicited on both sides, and the electrical reactions of the paralysed muscles are normal. The patient is unable to close either hand, and when he endeavours to grasp an object, the hand becomes hyperextended at the wrist, so that it is nearly at right angles to the forearm; the first phalanges are at the same time extended upon the metacarpal bones, while the second phalanges are flexed upon the first, and the first upon the second. The metacarpal bone of the thumb is abducted and drawn backwards, so that it is almost upon a level with the metacarpal bones of the fingers, and the phalanges are extended upon one another, and upon the metacarpal bone. The thenar and hypothenar eminences are flattened, the metacarpal bone of the index finger can be felt immediately under the skin, owing to the wasting of the abductor indicis, and when the mass of tissue which in health lies between the metacarpal bones of the thumb and index finger is grasped between the finger and thumb, it is felt to consist of scarcely anything more than a fold of skin, owing to the wasting of the abductor pollicis, and the flexor brevis pollicis.

The small muscles of the hands contract to a strong faradic current: they contract to the galvanic current with twenty-five cells Leclanché on negative closure, and with thirty cells on positive closure. There is incomplete anæsthesia of the skin of the lower extremities and trunk up to the level of the first rib, and the anæsthetic and sensitive areas merge into one another without being separated by a band of hyperæsthesia. The sense of temperature is almost entirely lost over the lower extremities and trunk, and two points have, on an average, to be removed four inches from each other before they are recognised as separate. The sense of pain as tested by pricking with a pin is not much diminished, but on pinching a fold of skin, the patient states that it feels numb; there is no perceptible retardation of sensibility, touch is localised fairly well, and the muscular sensibility is normal. The inner surfaces of the arm and forearm, and the inner half of the dorsal and palmar aspects of the hand and two and a half fingers, are also incompletely anæsthetic on both sides (Fig. 17). In these regions the patient can feel the prick of a pin fairly well, but he can bear strong pinching of the skin without giving evidence of pain. He cannot localise touch well in the anæsthetic areas, and two points have to be separated four inches before they are recognised as separate. The ordinary breathing of the patient is diaphragmatic, but he can still elevate the ribs by a voluntary effort, and the intercostal muscles are then seen to act. He has to strain a good deal before he urinates, but he has had no retention. The bowels are somewhat constipated. The pupils are widely dilated, but they react fairly well to light and accommodation. The optic discs are normal. All the internal organs are healthy.

April 11*th*, 1883.—The patient still lies on his back in bed, without having undergone much change since last report, although his general condition has on the whole improved. The right pupil is now considerably more contracted than the left, and the palpebral aperture is decidedly narrower on the right than on the left side.

Dec. 14*th*, 1883.—The patient's health had improved so much that he was transferred to the Cheadle Convalescent Hospital, about the middle of October last, and since that time a still further improvement has taken place. The anæsthesia has almost entirely disappeared; the small muscles of the hand have become plumper; the patient's grasp is good; and he can move his legs freely in bed, although muscular tension can still be provoked by passive movements of them, and the tendon reactions are somewhat exaggerated. The pupils are now equal, and there is no difference in the size of the palpebral fissures. The patient can raise himself in bed and sit up, but is yet unable to walk without support. There is no prominence or thickening of the spinous processes of any of the vertebræ, and no tenderness on percussion of any part of the vertebral column.

Fig. 17.

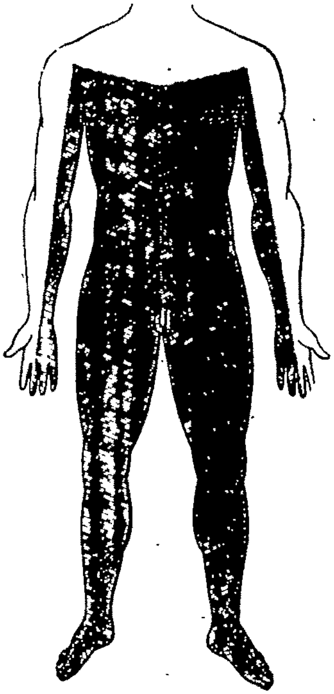

Fig. 18.

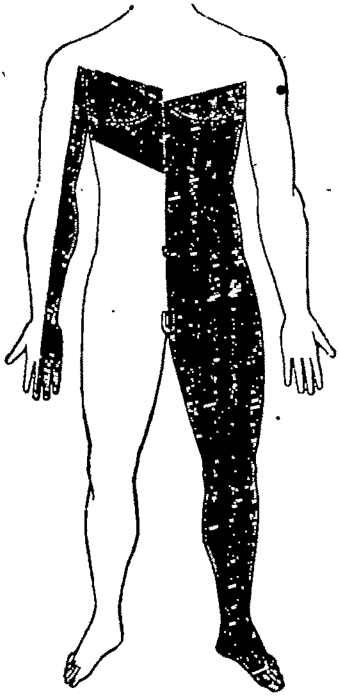

Fig. 17. The shaded portion shows the distribution of the anæsthesia in a case of cervical pachymeningitis, of which the lesion was situated on a level with the eighth cervical and first and second dorsal nerves.

Fig. 18. The shaded portion shows the distribution of the anæsthesia in a case of hæmatomyelia, in which the upper limit of the lesion was on a level with the eighth cervical nerve.

In this case, each hand assumed the peculiar claw-like form described by Charcot[1] as occurring in cervical pachymeningitis when the lesion is situated opposite the last cervical and first dorsal vertebra, and the distribution of the motor paralysis as well as the oculo-pupillary phenomena present in this case sufficiently indicated that the lesion was situated on a level with the junction of the cervical and dorsal regions of the cord.

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[1] See Charcot (J. M.), 'Leçons sur les maladies du système nerveux,' tome ii. 1877, p. 251.

The portion of skin which was anæsthetic in each upper extremity is supplied by the intercosto-humeral, the nerve of Wrisberg or small internal cutaneous, the internal cutaneous, and the sensory branches of the ulnar nerve. It is known from dissections that these nerves are derived from the eighth cervical, and first and second dorsal nerves, and these are the roots of the plexus which we would expect to have been involved in disease from the position of the lesion. It is scarcely necessary to add that the anæsthesia of the trunk and lower extremities were, in this case, caused by disease of the sensory conducting paths. In a case of hæmatomyelia recorded by myself,[1] the chief lesion was situated in the right lateral half of the spinal cord, and from the character of the motor, sensory and oculo-pupillary symptoms present it must have extended from the level of the ninth dorsal up to that of the eighth cervical nerve inclusive. The distribution of the anæsthesia is shown in Fig. 18. The anæsthesia of the inner half of the cutaneous surface of the upper extremity and of the belt which encircles the half of the thorax on the right side is to be explained by supposing that the hæmorrhage had destroyed on that side, either partially or wholly, the posterior grey horns on a level with the eighth cervical and upper nine dorsal nerves. The anæsthesia of the half of the trunk and lower extremity on the left side was doubtless caused by injury of the sensory conducting paths in their way through the cord after crossing over to the right side. If these cases do not teach us anything very new, they at least confirm the knowledge already acquired from dissections of the brachial plexus and experiments on animals, and they serve to illustrate in a striking manner the distribution of the anæsthesia caused by disease of the inferior roots of the brachial plexus and the second dorsal nerve.

In order to determine the distribution of the sensory disorders caused by disease of the superior roots of the brachial plexus, we naturally turn to the cases of combined paralysis of the deltoid, biceps, brachialis anticus, and supinator longus muscles first described by Erb,[1] and in which the lesion is most probably localised in the root derived from the fifth cervical nerve. In these cases patients have complained of formication and numbness in the outer surface of the arm and forearm, as well as in the thumb and index finger,[2] but the sensory disorders are never prominent and soon disappear. It would, therefore, appear that the fifth root of the brachial plexus supplies sensory fibres to the external cutaneous and radial branches of the musculo-spiral, as well as to the musculo-cutaneous and probably also the median nerves, but the absence of anæsthesia and the rapid disappearance of the paræsthesiæ in the area of distribution of these nerves seem to indicate, that all of them receive sensory fibres from other roots of the plexus as well.

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[1] Ross (J.), 'The Practitioner,' Sept. 1882, p. 172.

The following case of rupture of the brachial plexus, which was kindly sent to me by my friend Dr. Cowan, of Newchurch, may be worth reporting here, inasmuch as it will serve to illustrate the sensory disorders which are caused by rupture of all the roots of the brachial plexus with the exception of the communicating branch from the fourth nerve.

J. F., aged 17 years, entered the Manchester Royal Infirmary on December 2nd, 1882, under the care of Dr. Ross.

He never had any serious illness until six months before admission. Working in a cotton-mill, his left arm was caught between the strap and a revolving wheel. He was lifted from the ground and carried round by the wheel, until he fell on the opposite side. He was at first stunned by the fall, but on recovering he felt as if his right arm were dead; and it was found to be quite powerless, as it has since remained.

Present Condition.—The patient is a healthy-looking young man, fairly well built and free from organic disease of the internal organs. His left arm hangs powerless by his side when it is not held in a sling. All the muscles of the upper arm, forearm, and hand are completely paralysed and atrophied. The circumference of the left arm about its middle is 6½ inches, and of the right 8½ inches; and the circumference of the left forearm a little below the elbow is 7 inches, and of the right 9½ inches. The electrical reactions of all the nerves of the brachial plexus are lost. The faradic contractility of the affected muscles is also abolished, but the galvanic contractility is increased, while a stronger reaction is obtained from anodal than from cathodal closure. The latissimus dorsi and the lower two-thirds of the pectoralis major muscles are also paralysed and atrophied, but the upper third of the pectoralis major and the pectoralis minor as well as the internal and external rotators of the humerus, are unaffected. The serratus magnus on the left side is not paralysed, but when the paralysed arm is raised vertically by the observer and fixed in that position, the digitations of the muscle do not become so prominent on the left as on the right side during deep inspiration, and there appears also to be a slight degree of relative wasting of the left muscle, but the electrical reactions are normal. When the chest is at rest after the deepest expiration, the left half on a level with the nipple measures 14 inches and the right 14¾ inches; whilst after the deepest inspiration the left measures 14¾ inches, and the right 16½ inches. The patient can perform certain movements at the shoulder joint with the muscles that remain active, which might lead one to suppose, without careful examination, that the deltoid, triceps, and biceps possess a slight degree of motor power. By leaning his body well to the healthy side and somewhat backwards, he can raise the left arm above the horizontal level in a forward direction. While in this position he can extend the forearm upon the arm at pleasure. It is found, however, that the arm is raised partly by a powerful rotation of the scapula, during which the inferior angle is drawn well forwards and the superior external angle is well tilted upwards, and partly by the action of supra-spinatus muscle. Professor Alexander Ogston examined the patient along with me a few days ago, and both of us were convinced that there was no contraction of the deltoid during this movement. Extension at the elbow is produced by a powerful outward rotation of the humerus while the limb is held horizontally, this movement placing the forearm in such a position that it becomes extended by its own weight. And again, when the patient produces an inward rotation of the humerus the forearm becomes flexed by the action of gravity, and the hand moves towards and nearly reaches the patient's mouth.

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[1] Erb (W.), 'Verhandlungen des naturhistorischen Vereins zu Heidelberg,' 1875, s. 180; see also Ziemssen's 'Encyclopædia of the Practice of Medicine,' vol. xi., 1876, p. 561.

[2] See Hoedemaker (H. ten Cate), "Ueber die von Erb zuerst beschriebene combinirte Lähmungsform, an der oberen Extremität." 'Archiv für Psychiatrie und Nervenkrankheiten,' B. ix., 1879, s. 738.

Every form of sensibility is lost in the skin of the hand, and in that of the forearm, with the exception of a small portion adjoining the elbow in its posterior and internal aspects. The skin of the inferior half of the anterior surface of the arm is also completely anæsthetic; but the superior half of the anterior surface, the skin of the whole of the external, posterior, and internal surfaces of the arm is sensitive to the prick of a pin, to pinching, and to touch, although those portions of skin manifest a slight diminution in the appreciation of separate points as compared with the corresponding parts of the healthy arm. Separate points are discriminated on the inner surface of the left or diseased arm above the elbow at a distance of 3 mm., on the healthy arm at 2 mm.; on the left arm near the axilla at 6 mm., on the right at 4 mm.; on the left arm

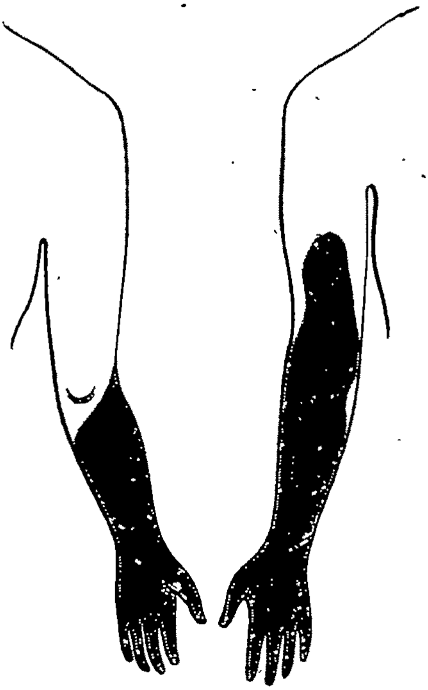

FIG. 19. FIG. 20.

Figs. 19 and 20. Posterior and anterior aspect of the arm respectively, in a case of rupture of the brachial plexus, the shaded area showing the distribution of the anæsthesia. By an oversight, the right arm is represented as being affected instead of the left.

over the shoulder at 5 mm., on the right at 2 mm.; on the posterior surface of the left forearm near the elbow at 2½ mm., and on the right at 1¾ m. The distribution of the area of complete anæsthesia is indicated by the shaded portions of Figs. 19 and 20.

The sensitive area and that of absolute anæsthesia are separated by a band of about an inch in vertical extent, in which pricking with a pin and strong pinching are slightly felt, but on the whole, the transition from one region to another is comparatively abrupt.

The skin in the anæsthetic area is thin and smooth; the affected extremity becomes cold, and of a dark blue colour on exposure; and the pulse is even more perceptible on the affected than on the healthy side, depending most probably upon a wasting of the tissues on the diseased side, which causes the artery to feel as if it lay almost immediately beneath a thin covering of skin.

The left pupil is smaller than the right in a diffused light, and the difference between them becomes greater when the eyes are shaded. The left pupil is dilated to a medium, and the right to a maximum degree of dilatation by atropine. The palpebral fissure is decidedly narrower, and the eye appears smaller on the left than on the right side. The colour of the face on the two sides is the same, and the temperature, taken in the external meatus of the ear, was found on an average of several observations to be ·8° Fah. higher on the left than on the right side; in one observation the temperature was slightly higher on the right than on the left side, and in the last observation made there was no difference between the two sides.

Dec. 10.—A quarter of a grain of pilocarpine was injected subcutaneously. In a few minutes the body was bathed in perspiration, with the exception of the paralysed arm which remained quite dry. The left half of the face perspired almost as freely as the right half.

Aug. 1883.—Through the kindness of Dr. Cowan, I was enabled to show the patient at the meeting of the British Medical Association at Liverpool, where he was examined, amongst others, by Dr. Broadbent.

In the case which has just been reported, the presence of complete paralysis with atrophy of all the small muscles of the hand and of the flexors of the forearm showed that the motor fibres of the inferior roots of the brachial plexus were ruptured, while the presence of a similar affection of the deltoid, biceps, brachialis anticus, and supinator longus showed that the motor fibres of the fifth root were severed, and consequently there are no grounds for believing that the intermediate roots derived from the sixth and seventh nerves had escaped, even although it is not accurately known what muscles are paralysed by isolated disease of one of those nerves. The absence, however, of paralysis of the inward and outward rotators of the humerus would seem to indicate that the motor fibres derived from the communicating branch to the brachial plexus from the fourth nerve had remained unaffected. But if the motor fibres of the roots of the plexus derived from the fifth, sixth, seventh, and eighth cervical, and first dorsal nerves were completely ruptured, it may be inferred that none of the sensory fibres of those roots had escaped, and the absence of electrical reactions in the affected muscles is totally opposed to the supposition that any reunion had taken place. The intercosto-humeral nerve was doubtless spared,[1] and it may be supposed to have conferred a degree of sensibility on the inner surface of the arm and a small part of the inner surface of the forearm which was only slightly below the normal as tested by a comparison with the corresponding parts of the healthy arm. The sensibility in the superior two-thirds of the anterior surface of the shoulder and arm, and on the whole of the external and posterior surfaces of the shoulder and arm as well as a small portion of the adjoining surface of the forearm was only slightly diminished as compared with corresponding parts of the healthy side, and sensation must have been conferred on these portions of skin by the sensory fibres of the communicating branch of the fourth nerve, and the sensory branches of the cervical plexus which descend over the shoulder.

In a case of excision of the brachial plexus by Maury and Duhring[2] for the relief of painful neuroma of skin, the sensations of pain and touch were absent, six months after the operation, over the whole of the skin of the forearm and hand as well as in that of the anterior aspect of the upper arm, as represented by the area included within the lines *a a*, and *b b*, Fig. 21. The whole of the posterior, external, and internal surfaces of the arm were sensitive to touch, and to the prick of a needle, the sensation being more acute in the superior than the inferior part of the arm. It will be seen that a rather larger portion of the skin of the anterior surface of the arm was rendered completely anæsthetic in this case, than in my case of rupture of the plexus. In excision of a portion of the plexus, the communicating branch from the fourth nerve would be divided, while in rupture it is most probably spared, and consequently in Maury and Duhring's case what remained of sensibility in the skin of the upper arm must have been conferred by the intercosto-humeral and the descending branches of the cervical plexus alone.

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[1] See Paget (J.). 'The Medical Times and Gazette,' March 26, 1864.

[2] Maury and Duhring. "Case of exsection of the brachial plexus of nerves for the relief of painful neuroma of the skin," 'The American Journal of the Medical Sciences,' vol. ii., 1874, p. 29.

Fig. 21.

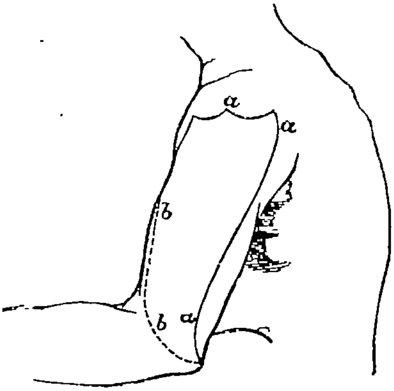

Fig. 21 (after Maury and Duhring). The line *a a a*, on the outer surface of the arm, marks the boundary above and outside of which sensation is preserved. The dotted line *b b*, marks the same for the inner surface of the arm.

The only cases which I have mentioned in this paper are those in which one or more of the cutaneous nerves of the brachial plexus had been completely divided, and in which the permanency of paralysis in the region of distribution of the corresponding motor fibres, or some other circumstance, had shown that no reunion of the divided ends could have taken place. From these cases the following conclusions may be drawn.

1. That one of the principal nerve trunks of the brachial plexus may be completely divided without giving rise to

complete anæsthesia in any part of the area of distribution of the sensory branches of the nerve; and that when complete anæsthesia does occur the portion of skin affected is very limited, and even the area of skin affected with partial anæsthesia is usually much less than that of the district corresponding to the anatomical distribution of the nerve.

2. That, as a general rule, the anæsthesia caused by division of one or more cutaneous nerves tends to become progressively less in degree and extent with the lapse of time. From this it follows that in division of one or more cutaneous nerves the area of normal sensibility tends to encroach upon the anæsthetic district, so that when one nerve (say the radial) is divided, its area of distribution, when judged by the extent of the anæsthesia, appears to be very small, as in Fig. 16, while when it is the only one of the three principal nerves of the hand that is spared, its area of distribution, as judged by the extent of the area of normal sensibility, appears to be unusually large, as in Fig. 9.

3. That the extent, degree, and even localisation of the anæsthesia caused by division of any nerve differ greatly in different cases, without our being able to discover anything in the nature of the injury to the nerve or in the external circumstances of the patient which would account for these differences (Figs. 15 and 16).

4. That the descriptions given by Krause and by Henle of the anatomical distribution of the digital branches, and especially of those derived from the median nerve, correspond more accurately with the results of pathological observation than those usually given in English anatomical works.

ON SOME PECULIARITIES CONNECTED WITH RETINAL IMAGES.

BY SYDNEY HODGES,

Associate of the Royal Academy.

In the 'Nineteenth Century' for October last, I detailed at considerable length some experiments I had been making in connection with the phenomena of after-images. It is not desirable to repeat my description of the experiments here. Those who are interested in the subject will have no difficulty in referring to them in the magazine in question. I wish, however, to recapitulate a few main points, by way of introduction to some physiological facts which will probably prove of more immediate interest to the readers of 'BRAIN.'

The main conclusions at which I arrived, after devoting several months to careful experiments, are the following:

1. That the colour of an after-image produced by gazing steadily at bright light or a strongly illuminated surface, such as white paper in sunlight, is not in any way dependent on the colour of the object we look at, but upon the amount of light thrown on the retina; either by the greater or lesser intensity of light in the object itself, or by the amount of time during which one looks at it.

2. That the succession of colours the image undergoes in forming and fading follows the order of the colours of the solar spectrum.

3. That the image is intermittent if the exciting object is looked at with one eye only, and intermittent in colour if it is looked at for a certain interval with one eye, and then for a similar interval with both.

These, briefly stated, are the main results of my observations. With reference to the first point, I may add that the best after-images are produced by placing a half-sheet of white note-paper in sunlight, and then fixing the gaze with absolute steadiness on a small pencil-mark in the centre. If the sun is shining brightly, and I look at a paper thus exposed for ten seconds and then close and cover the eyes, I get a blue after-image. If I look for fifteen seconds, I get a green image. If I look for twenty-five seconds, I get a yellow image; but no amount of exposure ever produces any colour beyond the yellow. With reference to the second point, if the eyes are kept closed, the colour of the image changes first to dull orange, then to pink, then to crimson, then to violet, and, after an interval, back to blue; thus, as I have before observed, following the order of the colours of the solar spectrum. After the image returns to blue, it darkens and fades altogether.

With regard to the third point, namely the intermission of the image if looked at with one eye only—which fact seems to be considered the most valuable discovery from a physiological and probably from a pathological point of view—I would remark that various experiments have led me to the conclusion that, *under certain circumstances*, one eye, if rigidly fixed, is incapable of a sustained impression of vision. The reasons among others, which have induced this belief are the following:

If I fix one eye on a certain point in the sky—a small white immovable cloud is the best object—and then close and cover the other eye with the hand, in about half a minute or a minute, if the eye is kept very steadily fixed, a dark cloud like London smoke comes over the point at which I am gazing, and entirely blots it out. After an interval of a few seconds the cloud reappears, and again, after an interval, disappears.

If I look at a sheet of paper in sunlight with one eye for some twenty seconds and then close and cover the eye, I get a coloured after-image which comes and goes at intervals of six or seven seconds. This image, I should remark, is not nearly so vivid as the images formed by looking with both eyes, and it is much longer in forming.

If I look at a white object through a glass of a certain colour with one eye and through a glass of another colour with the other eye at the same time, the colours do not mingle, but the object appears first of one colour and then of the other, alternating at intervals of a few seconds. This last experiment has been dealt with at some length by Helmholtz, but the former ones I have never seen recorded.

It appears to me that the explanation of the intermission lies in the close sympathy which exists between the two eyes, from the peculiar manner in which the optic nerves unite and again divide. Each must necessarily partake, to some extent, of the sensation conveyed to the other. For instance, after gazing at the white cloud for a certain time and receiving the impression of light in one eye, the darkness of the closed eye begins to assert itself, and insists on conveying the sensation of darkness to the sensorium before the eye exposed to light is again allowed to convey its impulse of light.

Since the publication of the article to which I have referred, I have discovered a few new facts in connection with the sensation of colour which seem to me of considerable interest. I must, however, admit that one of these is to some extent antagonistic to some theories I before advanced. In connection with the resources of the retina, I ventured to raise some objection to the "fatigue theory," so popular with Helmholtz and other physiologists. My idea was that the phenomena connected with this theory were due rather to a principle of compensation. The fatigue theory is founded chiefly on the fact, that if we look at a particular colour for a certain interval, and then remove the eyes to some plain surface, we see the complementary colour. I urged that this could hardly arise from fatigue, because if we continued looking at the original colour, although it might become less vivid, it never wholly disappeared. It appeared to me that, if it arose from actual fatigue of the rods and cones, which seem to be the seat of the primary sensation of vision, we should after a time fail to appreciate the colour altogether. This I now find, under certain circumstances, is actually the case. The simple experiment which led to my discovering this is, I think, new, and may excite some interest. It is of course known that if the three primary colours, or all the colours of the solar spectrum are painted on a disk, and the disk is made to revolve rapidly, the colours disappear and the disk assumes a greyish-white tint. I was anxious to ascertain if the sensation of white could also be obtained by prolonged exposure of the retina to the three primaries without the revolving process. I therefore coloured a piece of cardboard with faint tints of the three primaries accepted by artists, namely red, blue, and yellow, painting these colours in stripes of equal width. I then held the card in the sunlight, fixing my eyes upon a pencil mark in the centre. In a few seconds the colours began to grow fainter, then to merge into each other, then to disappear altogether, and it appeared to me as if I were holding a piece of white card in my hand. If, however, I moved the eyes to ever so small an extent, either to the right or left, so as to bring the different tints on to another part of the retina, they immediately reappeared, and were apparently intensified in tone. This experiment seemed to me a conclusive proof that if the three rays from the three primaries were conveyed to the retina simultaneously, the sensation of white was produced, thus, apparently, at the same time proving that red, blue and yellow were the primary constituents of white light.

I next proceeded to experiment with the three colours accepted by Dr. Thomas Young, Professor Helmholtz, and other physiologists, as the primaries, namely red, green, and violet; painting faint tints of each on a card in the same manner as before. Somewhat to my surprise the result was precisely the same. When held in sunlight, the colours disappeared if the eyes were immovably fixed on them. This led me to think that it must be something apart from the combination of particular rays which produced the sensation of white. I therefore tried the experiment with two colours. Again the result was the same. I then isolated the centre colours of the first card, by covering the other two colours with white cards. The result was still the same. The colour disappeared, and the three cards presented an almost uniform surface of white. I was thus forced to the conclusion, that it was not the combination of certain coloured rays which produced the sensation of white, but that the retina was incapable of sustaining the impression of strongly illuminated faint colours on the same portion of its surface for any length of time. As I have before remarked, if the gaze was shifted to ever so slight an extent, the colour at once reappeared; which fact leads me to the conclusion that it is only by the rapid and constant shifting of the eye that we retain the full sensation of colour. There have been so few recorded observations of the results obtained by fixing the eyes immovably on strongly illuminated objects, that this class of experiments seems to me to open quite a new field of investigation. The disappearance of the colours came upon me as a new and most surprising fact, and certainly does favour the theory of fatigue.[1] I should mention that the same effect may be produced, to a certain extent, by strong gaslight or ordinary daylight, but the result is not nearly so satisfactory.

I am anxious to record another very curious fact in connection with the intermission of the image before referred to. I happened to be looking at the sun through my telescope one morning, for the purpose of observing some sun-spots. On withdrawing my eye from the telescope, and turning towards a dark corner of the room, I found that I had a pink after-image of the sun. Remembering that it was the result of looking with one eye only, I anticipated the intermission, and found that it almost immediately commenced. I then took a rapid glance at the sun with both eyes, and then closed and covered them. The result was very curious. The intermittent after-image first produced was still coming and going at intervals of a few seconds, and through this intermittent image was seen the non-intermittent after-image produced by the momentary glance with both eyes. It should be borne in mind that the first image, having been obtained through the telescope, was magnified some eighty diameters, so that the second image was only seen as a small spot in its midst, but still it was seen in the *same field*, and therefore presents a most curious illustration of the extraordinary and complex powers and capabilities of the retina—the power to retain two distinct impressions of the same object upon the same spot.

Dr. Crichton-Browne seems to be under the impression, that in my former paper I did not make sufficient allowance for the personal factor. He considered that I possessed a special faculty for the production of after-images. This idea, however, is not borne out in my experiments with others. When the conditions are the same, the results have been almost identical, even with those who have experimented under my directions for the first time. Almost all the experiments, described both here and elsewhere, have been tried with others, and generally with the same results. I have no doubt, however, that the faculty of observing after-images may be greatly increased by cultivation. It is evident that the retina is never wholly at rest. Even in the middle of the darkest nights, in bed, with the eyes closed, one is conscious of certain colours passing in waves across the retina, and of flashes and rings of light resulting from sudden movements of the head. One very curious and very constant sensation is that of certain concentric rings, or waves of colour, which appear to flow from the outer circumference of the retina towards a variable disk of colours in the centre, generally of a crimson or purple tint. I have no reason, however, to suppose that even this sensation is abnormal.

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[1] Since this was written, Dr. Tuke has sent me a paper by an American writer which induces me to think that these effects are produced by changes in the visual purple.

NOTE ON MENTAL SUGGESTION.

BY PROF. CHARLES RICHET, M.D. (PARIS).

AMONG the various phenomena which have recently excited the curiosity of investigators, none are more interesting than those to which the name of "mental suggestion" has been given. It is true that these facts—supposing that they be facts—have long been affirmed by professional magnetisers; but none has hitherto been able to give a formal demonstration of it. The more indeed they sought to prove mental suggestion, the more it seemed to escape every scientific control.

Let us first define the term "mental suggestion."

Suggestion implies the inspiration of a thought to a hypnotised, magnetised, or normal subject. Whether this suggestion be the consequence of a gesture, word, or any other indication, it is a *material*, not a *mental* suggestion, as it were, easy of explanation, and not offering any cause for surprise. But what would be most important to prove is that this suggestion may be exercised, without the least external gesture, by the effort of thought alone. Such a suggestion, occurring independently of the least external indication, would be *mental.*

The following example will explain the difference between expressed and mental suggestion: Assuming the subject to be hypnotised and sensitive, it will be enough to describe a few zigzags with the finger on the floor, in order that the magnetised individual shall believe he sees a serpent. This is an example of expressed suggestion, and the production of the image *serpent* in the mind of the subject presents nothing wonderful, in presence of the cerebral excitation.

What would be highly astonishing is that the same image should arise without an external gesture or indication, and that the intellectual effort should, without any external manifestation, provoke the idea *serpent* in another mind. This is what many magnetisers say does take place, and it must be granted that some are of good faith, and that a few are even pretty good observers. But hitherto I am not aware that they have been able to prove the fact.

Hence he who should give a formal and scientific demonstration of mental suggestion would certainly make a true discovery, and one of the most important of our times. For the honour of a discovery does not belong so much to him who affirms a fact without demonstrating it, as to him who proves it after it has been affirmed by others. I have long sought, and am still seeking, to obtain a decisive proof of mental suggestion; unfortunately I must confess that my results have not been satisfactory, though I have had to do with some very sensitive and well-trained subjects.

My only object here is to indicate how the experiment may be performed. It is necessary, before all, that the action to be suggested mentally be simple, and not ridiculously complicated, as when the object is to deceive an ignorant public. The simplest way is to make the subject count aloud, and at any moment to give him a mental order to stop, after he has been warned that he is going to be stopped during the process of counting. In order to effect this, one must have sufficient self-command to restrain the least movement, or even the least change in the respiratory rhythm—in other words, to secure the purely mental nature of the suggestion. For the hyperæsthesia of the subject is sometimes such that the least gesture is perceived through the disturbance of the air it produces.

Following such a method, I have repeatedly ascertained that it is almost impossible to obtain a proof of mental suggestion—at any rate I have been unable to do so. It happens that the subject stops at the desired number, once or twice or three times, at the beginning of the experiment; but even this is not regular. At any rate, after the second answer the results become so divergent as to exclude every scientific deduction.

It has also seemed to me that there is a certain delay between the number mentally selected and the number at which the subject stopped. If, for instance, the number 8 is thought, the subject, counting aloud, stops at 11.

These data, however imperfect they are, and far from establishing the almost miraculous fact of mental suggestion, will perhaps serve to some more successful experimenter for giving a demonstration which hitherto has not been obtained.

A NEW HISTOLOGICAL METHOD FOR THE STUDY OF NERVE-TRACTS IN THE BRAIN AND SPINAL CHORD.

BY DR. SIGM. FREUD,

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In the course of my studies on the structure and development of the medulla oblongata I succeeded in working out the following method, which will be found a powerful aid in tracing the course of fibres in the central nervous system of the adult and the embryo.

Pieces of the organ are hardened in bichromate of potash, or in *Erlicki's* fluid (2½ parts of bichromate of potash and ½ of sulphate of copper to 100 parts of water), and the process of hardening is finished by placing the specimen in alcohol; thin sections are cut by means of a microtome and washed in distilled water. The washed sections are brought into an aqueous solution of chloride of gold (1 to 100) to which is added half or an equal volume of strong alcohol. This mixture is to be preferred to the simple aqueous solution of chloride of gold, which has been hitherto used in staining preparations; the sections are to remain in it from 3 to 5 hours. With the aid of a wooden rod (metal to be avoided) they are taken out of this solution, washed in distilled water and placed in a concentrated solution of caustic soda (1 to 5 or 6 of water); which very soon renders them transparent and slippery. After 2 or 3 minutes the preparations are taken out of the soda with the same wooden rod (toothpick or match), and the superfluous soda is allowed to drop off. The sections are then, with the soda they still contain, put into a 10 per cent. solution of iodide of potash where they almost immediately receive a tender rose-colouring, which changes into darker hues of red during the next 5 to 15 minutes. Now if the preparation be from the nervous system of the adult, it may be simply washed in water and transferred into alcohol, to be mounted in the usual way. But if it be of the brain or spinal cord of the new-born or the embryo, this treatment would spoil the section, by causing it to shrink, and throwing it into folds. Therefore these preparations must be brought upon a glass-slide by means of a camel's-hair brush, and dried by gently (without pressure) covering it with a piece of filter-paper. If the sections be very thin and soft, even this must be avoided; for the fibres of the paper would leave traces on the surface of the sections, and render them unfit for the study of the nervous elements. Nothing else can be done but to apply a piece of filter-paper to the edges of the stained preparation lying on the slide, and to draw off the alkaline fluid in this way. This is by far the most tedious stage of the process, yet it is always possible to avoid shrinking and to preserve the most sensitive preparations. The sections nearly dried are allowed to remain in water a few minutes, and are then treated in the way mentioned above.

This method will never fail (as all methods of staining by chloride of gold will do) if the specimen be not overhardened and brittle, as it is sure to be if kept in the hardening fluid an inordinate length of time. In order to stain the central nervous system of the adult, the method just described may be modified in many ways; but satisfactory preparations from the new-born or the embryo will require scrupulous attention to the details mentioned.

By this method the fibres are made to show in a pink, deep purple, blue or even black colour, and are brought distinctly into view, while the grey substance, vessels and neuroglia, lost in the slightly tinged background, are not obtruded upon the attention of the observer. A good many fine fibres, which cannot be revealed by carmine, and were not known until the methods of *Exner* and *Weigert* came into use, are seen scattered everywhere throughout the white and grey substance. In the adult the big nerve-cells also appear, and the ensemble of fibres is much too complicated for analysis; in the new-born and the embryo, the nerve-fibres alone are strikingly brought out, and those bundles, which are already possessed of a medullary sheath, are distinguished by darker colouring from the others. Examined under higher powers, the single axis-cylinders are so well defined as to enable one to count their number.

While this method is not adapted to the study of the grey substance, it is believed that it will prove of great service in the study of nerve-tracts, particularly if the central nervous system of the new-born or the embryo be made the subject of investigation.

Clinical Cases.

DESTRUCTION OF OCCIPITAL LOBE ACCOMPANIED BY BLINDNESS.

BY D. J. HAMILTON, M.B., F.R.S.E.,

Professor of Pathological Anatomy (Sir Erasmus Wilson Chair), Aberdeen University.

As lesions involving the occipital lobe, and that only, are somewhat rare, the present case, which is almost unrivalled for the isolated character of the lesion and the complete destruction it produced, may perhaps be of interest and value as tending to locate the function of the occipital region. The patient was under the care of Dr. James, in the Edinburgh Royal Infirmary. I am indebted to him for the notes of the history. The examination of the nerve-centres was made by the method recommended by me in this journal for July 1883, and I now possess a complete series of permanent preparations of entire horizontal sections of the brain, showing the points to which I refer in the following record. The lesion and its effects could be traced out almost diagrammatically, and the method of preparation presents this great advantage, that the parts can be retained for future reference and comparison.

The patient was a female, C. R., aged 36, a mill-worker by occupation. She was admitted to the Edinburgh Royal Infirmary on August 9th, 1882, and remained there until the time of her death. She was nearly six weeks under observation in the Institution.

History.—Patient's father died about six weeks ago of a fit, the nature of which she cannot recollect. Her mother is alive, but is not strong. She has always complained of her breathing. Patient has four brothers and two sisters, all of whom are quite healthy. She has always been able to take her food well, and does not drink at all. She leaves the house only when she is going to or coming from her work. She has never had any previous illness nor accident.

About six months ago she had great sickness and vomiting.

This seems to have been connected with the taking of food, and occurred at no special time of the day. She had no particular pain during vomiting. The vomited matter seems to have been chiefly composed of what she had been eating. It was neither frothy nor of a greenish tint. She went to a medical man in town, and he prescribed iron for her. This sickness lasted for about a month. About this time she also complained of having severe headaches. After this she was subject to what she says were fits. These did not occur every day; but she had occasionally more than one in one day. She was not in the habit of crying out, nor did she bite her tongue. She had no convulsions. During them she seems to have been quite unconscious of all her surroundings, and power of vision disappeared during them. They seem to have been more of the nature of faints. They have gradually gone away, and she has not had one for more than six weeks. All this time she has complained much of headache. She has always taken the medicine she got from the medical man. When these fits were gone, she complained of her eyesight. It has gradually been getting worse and worse, till now she is almost quite blind. There is an obscure history of a squint. She cannot say how long she has had it, but it has been mentioned to her several times by different people. She cannot say if it was constantly present, or merely coming on for a short time and then passing off again. When she was first taken ill the pain was in every part of her head, but now it is confined to the front of the head between her temples. During all her life she has been troubled with palpitation.

State on admission.—Patient is not very well developed. Her muscles are small, but firm. Her grasp of the hand is feeble. She lies quite motionless in bed with her legs usually drawn up. She has a very staring look. The pupils are widely dilated. There is a slight squint outwards of the right eye. She usually lies on her right side. There is no evidence of disease or previous injury on her person. She has an extremely anxious expression of face.

Circulatory System.—She has no pain in the region of her heart, but is much troubled with palpitation. When she walks quickly, or has anything to annoy her, she is troubled with shortness of breath. The præcordia is natural as regards form and shape. A strong impulse is visible between the 5th and 6th ribs in the mammillary line, but it is rather diffused. The impulse on being felt is of a powerful and thumping character. It is apparently followed by a much slighter impulse.

On percussion, the deep dulness is as follows:—The upper border is on a level with the 2nd left rib, the right border is in the level of the 3rd and 4th ribs, in the parasternal line, the apex-beat is slightly to the outer side of the mammillary line between the 5th and 6th ribs. In the mitral area, on auscultation, both sounds were found to be of natural rhythm, but are rather feeble. In the other areas the sounds are similar. The pulse at the wrist is feeble, small, easily compressible, but regular.

Respiratory System.—There are 24 respirations a minute. The breathing is quiet and regular. It is chiefly abdominal. She has a slight cough which does not trouble her much, except at night. There is no spit. The chest is fairly well formed. The costal angle is very acute. The interspaces are well marked. Owing to the great prominence of the 2nd rib there seems to be some flattening under the clavicles. The fremitus is not increased on either side, but is rather better marked on the right side under the clavicles, and is not felt on either side below the 2nd rib in front. There is considerable impairment on percussion on the left side throughout the front. On the right side there is fair resonance. At both apices (1st interspace) there is great prolongation of inspiration, and there is a pause between the two. It is more or less bronchial in character on both sides. On the right side this is much better marked than on the left. On the left side fine crepitations can be heard, and the vocal resonance is somewhat increased. In the 3rd interspace the same prolongation of expiration is noticed, but below this it is normal. Posteriorly vocal fremitus is better marked on the right than on the left side. Percussion is slightly impaired on the left side. At the left apex there is bronchial breathing with increased vocal resonance. At the right apex the character is much the same. There is an impaired percussion note in the scapular and interscapular regions to within an inch of the angles, and on the left side there is also some impairment in the upper part of the scapular region. In the interscapular region the breathing on the left side is rather feeble. On the right side it is natural. Over the left scapula expiration is prolonged and breathing is feeble. Fine crepitations are heard on forced inspiration. Over the right scapula the character is the same. At the left base the breath sounds are hardly audible at all, and the same is the case on the right side. Throughout the right axilla the breath sounds are feeble.

Nervous System.—Patient could not give an account of the origin of the blindness, except that it came on very rapidly about six months ago. There was nothing except dimness of vision at first, no diplopia or colour-blindness. On admission the patient was in a very torpid state, and did not answer clearly. She was able to distinguish light from darkness, but answered at random as to how many fingers were held up. Dr. Argyll Robertson reports that there is very marked optic neuritis in both eyes, and that the left is the more advanced.

Hearing is fairly good. There is no complaint of noises in the head. Taste is not acute, and smell is almost absent. No difference could be detected on the two sides. As to sensation and sensibility there are no abnormalities complained of, and the sense of touch is nowhere impaired. Tickling did not cause reflex movements. Organic reflexes were normal. Tendon reflex very slight. Abdominal skin reflexes were absent. As to the mental condition there were no delusions manifested, and patient gave a fair account of her past history, answering questions apparently correctly, but not volunteering any information; nothing abnormal discovered except her extreme torpidity, which persisted during her whole stay in hospital.

The torpidity grew worse, and she gradually sank on Sept. 18th, 1882, the symptoms having been present for six months.

It should be mentioned that the temperature remained normal up till within a few days before death, and even then the highest attained was 101·2 F.

The post-mortem examination was made by me on September 18th, 1882.

The result of it was the following :—

External Appearances.—Height, 60 inches. Circumference at shoulders, 29¾ inches. The rigor-mortis was fairly marked. The whole upper surface, as well as the dependent parts, presented an anæmic appearance. The pupils were both considerably dilated, and of equal size. The fingers were slightly clubbed.

Heart.—The aortic valve was competent and the organ otherwise apparently healthy.

Lungs.—Right weighed 16 oz. At the apex there was a small cicatrix with a slight cheesy deposit. The lung otherwise was healthy. The left weighed 1 lb. 4 oz. Within the upper lobe were two cavities; one at the apex about the size of a small orange, the other, somewhat smaller, towards its base. They were not ordinary phthisical cavities, but had more the characters of gangrene. The uppermost cavity had a strong gangrenous odour. Their edges were infiltrated with some solid tissue, apparently cancerous, which also extended into the bronchial glands. There had apparently been a recent hæmorrhage into the lower cavity. The lower lobe was congested, but was otherwise healthy. Microscopically the tumour was composed of large flat cells, evidently cancerous in their nature.

Liver.—Weighed 2 lb. 11 oz. Slightly congested.

Spleen.—Weighed 4 oz. Healthy.

Kidneys.—Left weighed 4½ oz. Slightly congested. The right was in a similar condition.

Stomach.—The mucous membrane was slightly congested.

The Brain weighed 3 lbs. The skull was particularly thin. The surface of the hemispheres was specially dry, and the convolutions were flattened. Occupying the occipital lobe on the left side, and entirely confined to it, there was a tumour apparently growing from the dura mater. The dura mater was closely and inseparably adherent. The tumour, so far as could be made out from superficial examination, seemed to occupy the whole of the occipital lobe. The parietal lobe was intact. The optic nerves and tracts did not appear to have undergone marked atrophy. The dura mater was easily separated from the skull.

Eyes.—Both discs were much atrophied, and the retina for some distance round them had a yellowish tint.

The brain was carefully hardened and prepared as before mentioned. The following was the result of the detailed examination.

The tumour was about the size of a billiard-ball. It was firm in consistence, and could not easily be dislodged from the brain tissue, with which it appeared to be continuous. Microscopically it presented the features of a flat-celled epithelioma. The cells were quite characteristic, and were evidently growing, as the nuclei in all of them were particularly large and prominent. They were arranged in characteristic cell-nests in some parts, and in others they had a concentric laminated appearance round the blood-vessels. The tumour was vascular, the vessels being in many cases tortuous and distended.

Round about it there was a zone of redness, about one and a half line thick, which, on microscopic examination, proved to be caused by great numbers of distended and extremely tortuous capillary blood-vessels. The tissue in this zone was made up of disintegrated brain substance. Great numbers of compound granular corpuscles could be seen, evidently resulting from the fatty degeneration of the nerve and connective tissue elements. The tumour had not pushed the brain tissue aside, but appeared to have been incorporated with it, or at any rate, to have given rise to complete destruction of it throughout the whole occipital lobe. It involved both the grey and white matter, extending forwards with the latter for a distance of something like an inch to an inch and a quarter. On the supposition that the grey matter of the occipital lobe had been simply pushed inwards by the pressure of the growth, I made a very careful examination of the surrounding parts for the presence of nerve cells or any remnant of the cortical substance. Nothing of the kind, however, was discoverable, and I am therefore strongly inclined to believe that the grey matter of the cortex was entirely destroyed. The amount of disintegration in the red zone above described as surrounding the tumour, entirely precluded the idea of the cortical substance having been merely inverted or pushed aside. The occipital lobe in fact, in the preparations which I possess, appears to have been quite cleanly cut out, and its place substituted by the tumour mass. It was the only tumour found throughout the central nervous system, and it seems probable that it was secondary to the gangrenous neoplasm contained in the left lung.

Such was the condition of the tumour and its immediate neighbourhood, and although it was the only tumour contained in the brain, it must not be supposed that it was the only lesion. I naturally looked at first for some trophic effect upon the nerve fibres coming from the destroyed area. I question, however, whether anything of the nature of a secondary degeneration had occurred.

It will be remembered that the fibres running back to the occipital lobe are not diffusely spread out in the parietal and occipital white matter, but are confined to a ribbon-like band which lies closely in contact with the tapetum or callosal fibres lining the posterior cornu of the lateral ventricle. The borders of this band are perfectly defined. There is no mixing up of the fibres in an indiscriminate manner with those derived from the cortex proper of the parietal lobe and the posterior extremity of the ascending parietal convolution. The callosal fibres of the tapetum no doubt run through it, and there is an intercrossing of the two sets, but the margin of those coming backwards is perfectly isolated.

Gratiolet[1] was the first to describe this band, and consequently it is known at the present day usually by the name of "Gratiolet's optic radiation." He supposed that he had traced its direct connection with the optic tract.

Now this "optic radiation" contains, in fact, all the fibres going to the occipital lobe, unless those which join the corpus callosum. The latter run across the "optic radiation" and enter the tapetum, to be by it conveyed still further forwards to the corpus callosum. The fibres composing it belong to many different sets, and undoubtedly are possessed of widely diverse functions. So far as I can make out, they are the following:—

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[1] "Anatomie comparée du Système Nerveux." 'Comptes Rendus,' 1854, t. xxxix. p. 274.

1. A very large band which connects the Island of Reil with the tip of the occipital lobe. It runs directly backwards, and can be seen with the greatest ease in a section made in a horizontal plane. It is derived, in front, from the *unnamed white substance of the Island.*
2. A large band of fibres, supposed to be sensory in its function, which turns directly into the posterior third of the posterior division of the inner capsule.
3. Fibres which directly enter the optic tract of the same side.
4. A band to the corpus geniculatum internum.
5. Another to the corpus geniculatum externum.
6. A third to the pulvinar.
7. Fibres to the thalamus itself.

These all run directly back to the occipital region in a single band; and although this band does become smaller as it passes backwards, yet by far the greater part of its fibres end in the occipital cortex. In fact, the occipital lobe is made up of the expansion of this band and the fibres which enter the corpus callosum.

If, therefore, a secondary degeneration were to occur it would in all probability be located in the "optic radiation," or in the tapetum. My attention was accordingly directed to the examination of these, with the following results. Immediately on the same plane as the centre of the tumour, the "optic radiation" showed some amount of disintegration. It could be seen with the naked eye, because of the degenerated part being clearer than the surroundings. The degeneration was situated in the middle of the "optic radiation," and was noticed in only a few preparations. The greater extent of this occipital band was perfectly free from anything resembling a secondary trophic lesion. On examining the degenerated part microscopically, it was found to contain compound granular corpuscles, and the nerve fibres in it were destroyed. It ran forwards for a distance of about an inch and a half, and then seemed to terminate. In case the degenerated fibres had passed into a different plane, I examined sections above and below, but in none of them could I find any trace of the continuation of the degenerated fibres. I am strongly inclined to believe that the degeneration terminated here, at least in most of the fibres.

The question comes to be, was this destruction of the central portion of the "optic radiation" a truly trophic one, or was it a continuation forwards of the inflammatory zone round the tumour? The fibres which degenerate secondarily in lesions of the cortical grey matter are essentially those which are motor in their function. This is strongly insisted upon by Charcot, and it is a fact which is now generally recognised, that while a destructive lesion situated in the neighbourhood of the fissure of Rolando, will almost certainly, in course of time, give rise to secondary descending degeneration in the motor fibres derived from that neighbourhood, similar lesions may be located in the prefrontal area without causing anything of this kind. Very much the same thing appears to hold good of the occipital region, and I am strongly persuaded that the greater part of the destruction of the "optic radiation," above referred to, in my case was purely inflammatory. The vessels within it were all distended, and there were other evidences of the destruction of the nerve fibres having been brought about by causes other than trophic influence. I will not say, in the absence of clear proof to the contrary, that the direct fibres entering *the optic tract* from the "optic radiation" did not so degenerate, but I think, as regards the other bundles which compose it, there was no evidence of a progressive secondary trophic destruction of their fibres.

While, however, the destruction of the occipital lobe was unaccompanied by distinct evidence of secondary degeneration in the fibres springing from it, the lesion had given rise to widespread secondary changes of quite another description. These mainly consisted in an enormous enlargement of the cerebral hemisphere on the same (left) side as the tumour, while the right hemisphere remained of natural dimensions. In the preparations which were made through a plane corresponding with the centre of the tumour, the left cerebral hemisphere was at least twice as large as the right. The enlargement was universal, the frontal region being increased in size in the same manner as the parietal. The enlargement, however, was greatest in the parietal, although the proportional difference between that of the frontal and parietal regions was not very marked. The right side of the brain did not seem to have suffered at all, with the exception of a slight congestion of the occipital lobe, and, so far as could be seen, looked quite normal. The corpus callosum was thickened on the left side, not markedly on the right, and it was curiously twisted, owing to the displacement caused by the increased bulk of the diseased hemisphere. The lateral ventricles were also much twisted, and the left was in parts almost obliterated, from the protrusion into it of the mass of the neighbouring hemisphere.

The grey matter of the diseased hemisphere did not seem to have undergone much alteration in size. It was of almost exactly the same size as that in the other. Nor did its texture appear to be altered, the increase in bulk being almost entirely confined to the white matter of the centrum ovale and fibres coming from it. The white matter of the temporo-sphenoidal lobe had undergone the same enlargement that other parts of the corresponding hemisphere had.

The affected hemisphere presented an unnatural toughness. It was homogeneous when examined in a preparation clarified by my method, and the bundles of fibres, such as those of the inner capsule, did not seem to be at all destroyed, although they were separated by a much greater mass of intermediate tissue than is commonly met with. Those at the genu of the inner capsule were even more distinct than usual, evidently on account of their separation by intermediate tissue. As regards the basal ganglia, they appeared to have sustained very little injury. The bundles of fibres running into them were thickened, and this gave to the thalamus, at least, an appearance of enlargement. The grey matter, however, was unaltered, and did not seem to be swollen like the white. In fact, the increase in size seemed to have been exclusively confined to the white matter, any alteration in the grey being rather that of diminution in bulk from the pressure exerted upon it by the former. The thalami were considerably more congested than normally and equally so.

When the cause of this enlargement was investigated microscopically, it became evident at once that it was located in the connective tissue, not in the nerve elements. In preparations examined in Farrant's solution, a dense plexus of fibres was seen lying in the situation of the neuroglia, with here and there nuclei or glia-cells, as they might be termed, lying among them. The real character, however, of this newly-formed neuroglia was brought out only when the preparation was *half clarified* in oil of cloves, and mounted in gum damar solution. It then became manifest that the whole of this plexus of fibres and nuclei was resolvable into a dense mass of so-called "spider" or Deiters' cells. The appearance was exactly like that of a glioma I previously described in the 'Journal of Anatomy and Physiology' (vol. xiv.). The fibres were in reality enormous leashes of processes, which came off from the side of flat, many nucleated, connective-tissue corpuscles. They ran off in all directions, and, by interlacing, produced the appearance of a dense fibrous plexus. It was the growth of these that had caused the enlargement of the hemisphere.

Curiously enough, the nerve fibres had evidently not undergone degeneration to any very great extent, although they lay imbedded in this connective tissue, and surrounded by it on all sides. Strands of them could be seen running through the dense plexus of cell processes without any discontinuity or other evidence of destruction.

The optic nerves were examined with great care. In both, the fibrous septa running in from the pia-sheath were all considerably thickened and highly nucleated. In the left, however, this condition was very much better marked than in the right, and more so in one locality than in others. The particular locality to which I refer lay near the surface of the nerve (I cannot say which part), and occupied about one-third of the circumference. The septa were all much more thickened here than elsewhere, and more densely nucleated than in other parts. The nerve bundles contained in them were decidedly altered. The normal bundles of nerve fibrils, when seen on cross section in an unclarified preparation, are almost quite opaque. In the bundles enclosed within these thickened septa they had become more transparent than usual. They had a shrunken appearance, and when highly magnified the fibrils were seen to be disintegrated and in great part destroyed. They did not, however, contain compound granular corpuscles, nor were the intermediate nuclei more abundant than usual. Minute oil globules were, however, occasionally present in them. The other fibres did not present any of these appearances, and, with the exception of the thickening of the fibrous septa, the nerve seemed to be normal. The tract of degeneration above described was found in the left nerve only; the fibres in the right nerve seemed to be quite healthy. The vessels of the optic nerves did not show any evidence of distension nor of congestion. The small arteries in the pia sheath had a thicker middle coat than usual, but there was no evidence of undue vascularity. The subdural and subarachnoid spaces of the nerve were not in any way altered.

The corpora quadrigemina, cerebellum, medulla oblongata, and cord were all carefully examined, but no very particular lesion was found in them. The central canal of the cord was nearly obliterated by desquamated epithelium.

A FATAL CASE OF CONCUSSION OF THE SPINAL CORD.

BY SEYMOUR J. SHARKEY, M.B.,

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E. A., æt. 25, was admitted into St. Thomas's Hospital, under the care of Mr. Francis Mason, on January 27th, 1883, and died on March 12th, 1883. I have to thank my colleague for permission to publish the following account:

The patient was one of six persons upon whom a house fell; she was found with only her head protruding from a large heap of bricks and rubbish, and was brought on a stretcher to the hospital.

On admission, she was said to have been struck upon the back-bone, and she complained of extreme pain across the sacrum and of numbness over the left buttock. There was no anæsthesia and no paraplegia, nor was there any external evidence of fracture or of other gross injury. There was, however, retention of urine.

An abnormal condition of the right leg and arm was present, but this had existed from birth. It consisted of partial paralysis and atrophy of both limbs without loss of sensation, but with some contraction of the flexor muscles of the arm. The patellar and plantar reflexes were exaggerated on this side, but there was no ankle clonus.

During the first week she was in the hospital, there was complete retention of urine and the bowels only acted once, and that after an enema. After this, incontinence of urine and fæces set in, and persisted till death.

At the same time that the evacuations began to pass involuntarily, a bedsore suddenly appeared on each buttock. They occurred first as blebs situated on the lower edges of the buttocks, one on each side of the perinæum. They were perfectly symmetrical, and, as the patient lay on her back, they were quite free from contact with the bedding. They soon burst, and then became ulcers. About a week later a swelling made its appearance on the sacrum, which was angry-looking and inflamed, and rapidly became an open bedsore.

The temperature up to the time of the incontinence of urine was only slightly raised, generally ranging at its highest between 100° and 101° F.; but after this, up to the time of the patient's death, it was more elevated and of the hectic type, varying from normal to about 103°. This was accompanied by an ammoniacal condition of the urine and other signs of cystitis.

She died gradually of exhaustion on March 12th, and during the last weeks of life the acute bedsores began to heal.

Post-mortem examination.—Body emaciated, legs wasted, the right more so than the left; right arm also wasted and flexed at the elbow. The diminution in size of the right arm and leg appeared to affect the limbs *en masse*, and not to be due to wasting of any particular muscle. There were three bedsores, one of which was over the sacrum. The other two were remarkably and symmetrically situated. As the body lay on the table and the observer stood at the feet, two circular sores, about 1½ inch in diameter, could be seen on the buttocks, so situated one on each side of the perinæum, that they nowhere came in contact with the table. They were evidently healing, as a margin of pinkish-white, transparent, new skin, about ¼ inch broad, surrounded them.

There was no evidence of gross injury anywhere, either in the skull, vertebral column, muscles, ligaments, or long bones.

There was acute cystitis with thickening and contraction of the walls of the bladder, and likewise acute inflammation and dilatation of both ureters and thickening of their walls. The inflammatory process extended into the pelvis and calyces of the kidneys, and then in streaks through the medullary portion of the organs to reach the cortex, where the streaks ran into irregular groups of suppurating points. Both ureters contained pus, the right in the upper half of its course towards the kidney containing more than the left. This inequality seemed to be due to the presence of a slight stricture in the right ureter, halfway between the bladder and the kidney. The heart, lungs, and other viscera were normal.

On opening the spinal canal no fracture of bone, thickening of membranes, or hæmorrhage was observed; all appeared normal. In its lower half the cord was normally firm, except two or three inches in the upper lumbar region, where it was soft and slightly wrinkled transversely. The upper half, though not evidently pathologically softened, was not so firm as the lower lumbar region. In several places the right anterior cornu looked smaller than the left.

The brain was unsymmetrical, the whole of the left hemisphere being smaller than the right, and the temporo-sphenoidal lobe and angular gyrus more seriously malformed. This was evidently a congenital abnormality, and it has been fully described by me in a paper read before the Medico-Chirurgical Society on the 8th of May, 1883.

On microscopic examination of the cord sclerosis of the right lateral column was found, which was evidently in connection with the abnormality of the left cerebral hemisphere. The changes found in the softened portion of the lumbar region were distension of the perivascular spaces, and a soft, porous condition of the grey matter adjacent to the grey commissure on each side, but there were no distinct traces of inflammation.

Microscopic examination of the lumbar enlargement revealed nothing abnormal, except a vessel here and there with a few leucocytes around it.

Remarks.—With regard to the spinal cord the autopsy must be considered to have yielded negative results. For the softening in the lumbar region cannot be taken as evidence of disease, this being the position in which post-mortem softening is most frequently found; and of the latter process this cord probably afforded a specimen.

Nor did the microscope furnish any positive proofs, though it did some suggestions, that recent disease had been at work, and that the cord had all but completely recovered by the time the renal affection proved fatal.

If by concussion of the spinal cord be meant disease of the spinal cord set up directly by the shake of an accident, and not indirectly by the pressure of a fractured or dislocated bone, or of a hæmorrhage external to it, or by any such primary result of the accident, then it seems to us that the present case affords an example of concussion of the spinal cord. As no injury to bones, muscles, ligaments, &c., was detected on careful examination, we must assume that if the symptoms proceeded from disease of the spinal cord at all, the latter resulted directly from the force of the accident, whatever may have been the precise mechanism by which the result was produced.

The question then is, Was this a case of disease of the spinal cord?

It is true there was an absence of any loss of muscular power or of sensation, which are the usual phenomena produced by affections of this part of the central nervous system; but, on the other hand, other symptoms were present which are very common concomitants of paralysis and anæsthesia in acute disease of the cord, viz. (1) Retention and then incontinence of the evacuations, and (2) so-called acute bedsores.

These conditions must have been due either to direct local injury of the parts affected or to nerve lesions, either peripheral or central. We may set aside the idea of local injury of the bladder, bowel, and perinæum, as there never was the slightest evidence of it. The question only remains whether the symptoms were due to peripheral or to central nerve lesion. It is difficult to suppose that the nerves supplying the bladder and rectum should have been directly injured, when at the post-mortem examination not the slightest damage to other parts could be discovered; and it would be still more incredible that a precisely symmetrical lesion should have affected the perineal nerves. The only reasonable explanation of the exact symmetry of the acute bedsores in the perineum, which were situated on parts not exposed to pressure, is that the cord was diseased at the spot in the lumbar region where the perineal nerves approach or meet each other. And this would equally explain the affection of the bladder and rectum.

But it may be objected that no disease of the spinal cord was actually found. To this we answer, that physiological reasoning in this case is of more value than arguments based on our inability to find pathological changes, and that for two reasons:

1. Because it is impossible to examine more than a small portion of the cord microscopically. And although such scant examination shows us the anatomical changes of disease when extensive, yet our not finding alterations in the few sections we examine is no proof of the absence of a small focus of disease. In the present instance the focus must have been very limited in extent.

2. It is legitimate to suppose, that if we had had an opportunity of examining the cord a week or so after the injury we might have found disease; and yet that six weeks after the accident all morbid changes, except the few doubtful ones recorded, might have cleared up. For the bedsores had long been healing, and death was the result not of progressive cord disease, but of an unfortunate cystitis and suppurative nephritis which early accompanied the paralysis of bladder.

It seems then that the only reasonable conclusion is, that owing to the concussion of the spinal cord produced by the force of the accident a small focus of disease resulted, which would probably have ended in the recovery of the patient, had not secondary disease of bladder and kidneys unfortunately proved fatal.

Satisfactorily proved cases of concussion of the spinal cord are undoubtedly rare; but we must remember, that in order to prove them we require a post-mortem examination. This case at least suggests what other considerations would render probable, that in severe accidents small focal lesions may result from concussion, and although they may cause very obscure and annoying symptoms, yet as they are small in extent and have not damaged vital parts they terminate in recovery, and leave us to argue as to the cause of the clinical phenomena we observe, without giving us the opportunity of proving whether our conclusions are correct.

CASE OF CONCUSSION AND INFLAMMATION OF SPINAL CORD FROM GUNSHOT-WOUND OF BACK.

BY WALTER EDMUNDS, M.D., F.R.C.S.

On October 29th, 1883, A. B., aged 19, was shot in the back; he fell immediately, and had to be carried out of action. The surgeon who first examined the case reported, "he was found to be suffering from a gunshot-wound, the projectile, apparently a large slug, having entered obliquely near the inferior angle of the right scapula; the wound was large, its edges being inverted, contused and irregular. A careful examination was made; but the finger failed to discover any foreign body, the projectile having taken a tortuous course towards the dorsal vertebræ, where it is probably lodged. There is little doubt that the spinal cord has been severely injured, for there is complete paralysis below the seat of injury."

Patient was sent home, and on coming under our observation (Nov. 25th) the wound was seen to be on the level of the fifth dorsal spine and about an inch to its right. Probe would only enter about half an inch. He was completely paraplegic, and without any sensation below the level of the wound. No control over bladder or rectum. Both arms very weak, the right weaker than the left. Some cystitis. A bedsore over sacrum. He died March 5, 1884, four and a half months after receiving the wound.

At autopsy, the bullet wound had healed; no fracture, or indication of fracture, or dislocation of vertebra could be found, nor any track of the projectile. Theca vertebralis intact. The cord was seen to be much atrophied and softened about the level of the wound. The bullet could not be found, although carefully looked for. Kidneys hypertrophied: four or five ounces of serum in pericardium.

On hardening the cord in Müller's fluid, it was seen that there was universal myelitis and softening for about two inches opposite the wound; this gradually passing below into the sclerosis of the lateral and anterior pyramidal tracts, and above into sclerosis of the posterior median columns.

There was no indication of hæmorrhage, either external to or into the substance of the cord. It surface was uninjured.

Remarks.—Although the surgeon who first examined the case was no doubt justified in his opinion, yet the subsequent progress of the case ought to have thrown some doubt on the diagnosis.

The length of time (4½ months) the patient survived must be most exceptional in fracture of vertebra with direct injury to the cord.

Lidell,[1] speaking from an experience acquired in the American Civil War, says, "Leaving out of calculation such fractures as involve the spinous processes alone, the writer has never seen a case of gunshot fracture of a vertebra get well, and he might add he has never seen life prolonged for a month after the infliction of that injury."

And further, in a fracture of bone with the bullet imbedded in it, it would be surprising that the sinus should completely close and without the discharge of spicules of any bone.

Somewhat similar cases are to be found on record. Dr. Charlton Bastian[2] relates the case of a man who fell in his sleep from the top of a haystack 25 feet high: his legs were at once paralysed, and after suffering from spinal symptoms for six months, he died. At the post-mortem examination no changes whatever were seen, for the important changes which did exist in the cord were only rendered visible by keeping it for some days in a hardening solution.

Sir William Gull records a case due to an injury to the back of the neck, terminating fatally in thirty-four hours. At the post-mortem examination no fracture or dislocation was found, but a contusion of the cord at the level of the 4th and 5th cervical vertebræ.[3]

Leyden[4] mentions a case which he saw, in which concussion of the spine causing paralysis proved fatal in five days. At the post-mortem nothing whatever could be found abnormal, either microscopically or macroscopically.

In conclusion, it may be remarked that the myelitis which was found in this case could not have accounted for the paraplegia which immediately followed the injury. The term "concussion" has been used to indicate the cause of this.

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[1] Lidell, "On Injuries of the Spine, including Concussion of the Spinal Cord;" 'Amer. Journal of Med. Sciences,' 1864.

[2] 'Transactions of Med.-Chir. Society,' vol. l., 1867.

[3] Guy's Hospital Reports, 1858.

[4] 'Klinik der Rückenmarks-Krankheiten,' vol. ii., 1875.

Critical Digest.

ON THE SO-CALLED HEREDITARY ATAXIA, FIRST DESCRIBED BY FRIEDREICH.

BY J. A. ORMEROD, M.D.

REFERENCES TO PUBLISHED CASES:—

Friedreich's writings on the subject will be found in Virchow's Archiv, Bd. 26, 27, 68, 70.

Schulze gives the morbid anatomy of one of Friedreich's cases in Virchow's Archiv, Bd. 79.

Other cases are given by:—

Carré. 'De l'Ataxie locomotrice progressive.' (Thèse de Paris.)
Brousse. 'De l'Ataxie héréditaire.' (Paris, 1882.)
Kahler und Pick. 'Archiv für Psychiatrie,' &c., Bd. 8.
Seeligmüller. 'Archiv für Psychiatrie,' &c., Bd. 10.
Rutimeyer. 'Virchow's Archiv,' Bd. 91.
Schmid. 'Correspondenzblatt für Schweizer Aerzte.' 1880.
Carpenter. Medical Society of London, 1871.
Dreschfeld. Manchester and Liverpool Hospital Reports, 1876.
Gowers. 'Transactions of Clinical Society.' Vol. xiv.
D'Arcy Power. St. Bartholomew's Hospital Reports, 1882.
Kellogg. 'Archives of Electrology and Neurology.' Vol. ii.
Hammond. 'Journal of Nervous and Mental Disease," 1882.

FRIEDREICH, after describing the six cases first observed by him, gives the following general description of the disease:—

"There exists a chronic inflammatory degeneration of the spinal cord, leading to atrophy; which develops chiefly at the period of puberty, under the influence of hereditary predisposition; which is essentially limited to the posterior columns of the cord, begins in the lumbar region, spreads from thence upwards and downwards, and finds its limit in the medulla oblongata, after involving the trunks and nuclei of the hypoglossal nerve. . . .

"The clinical characters of the disease are impairment in the co-ordination and harmony of movements, developing gradually and spreading from the lower to the upper half of the body, and always involving finally the organs of speech; sensibility and the functions of the special senses, and of the brain, being intact. Paralysis of the sphincters and trophic disturbances are absent.

Less constant phenomena are curvature of the spine, sensations of vertigo, and nystagmus.

"From a clinical point of view we must regard the disease as a progressive paralysis of the faculty of the combination of movements; from the point of view of pathological anatomy, as a chronic degenerative atrophy of the posterior columns of the cord."

We shall give an abstract of two of his earlier cases, which possess the advantage of having been observed by him over a long period of time.

1. Charlotte Lotsch was one of a family of five children. Three were healthy, one (a brother) had the same disease as herself. She was well till 18, then, without assignable cause, weakness began first in the left leg, then in the right. This became worse in six years' time after a confinement; she now needed support when walking. At 26, weakness in both upper limbs appeared, and difficulty of speech. She was first seen by Friedreich at the age of 31, having been ill thirteen years. She was a spare muscular woman, yet unable to walk or stand without support. When lying down, she could execute simple movements of the legs, though with considerable effort. Simple movements of the hands and arms were executed easily, but finer movements, such as picking up a small object, threading a needle, &c., with very great difficulty. Her speech was thick and stammering. Sensibility in all its modes was normal, as was also the electro-contractility of the muscles.

A year later she had a sudden attack of diabetes insipidus, accompanied with a rise in the pulse-rate, flushings, and erythematous eruptions. These disappeared equally suddenly in three and a half months' time; transient salivation followed, also sweatings, diarrhœa and vomiting. The ataxia and the speech became worse, troublesome vertigo and palpitation set in. Before leaving the hospital (which she did in three years' time), the motor power had diminished in the lower limbs, the electro-contractility of their muscles was impaired; contracture of the feet and slight spinal curvature were noted.

She again came under observation at the age of 49, in the 31st year of the disease. In the interval she had experienced frequent cramps and pains in the lower limbs, and occasional difficulty of micturition, and she had lost power in the legs. Her state was now as follows. The lower limbs were paralysed almost completely, the muscles of the legs were flaccid, but there was frequently contracture of the thigh muscles. Sensibility was considerably diminished; the plantar reflexes were present, the patellar tendon reflexes absent. In the upper limbs the muscular power and cutaneous sensibility were unimpaired, but the incoordination had increased. There was well-marked curvature (angular and lateral) of the spine; she sat all hunched together, the head exhibiting oscillatory movements like those seen in a person going to sleep upright. The speech was almost unintelligible, nystagmus had developed; though there was no squint, and the action of the pupils was normal.

Agan there occurred a transitory elevation of the pulse-rate. She died of bronchitis, age 49, in the 33rd year of the spinal disease.

The post-mortem we shall give afterwards.

2. Friedrich Süss belonged to a family of five children; only one of them was healthy, three others were diseased like himself. At 15, he became affected with a weakness of the left leg, which in eighteen months spread to the left arm. Soon the right leg and arm became similarly affected. He had feelings of weakness in the back and loins. At 18, difficulty of speech showed itself. On rare occasions he had numb feelings in the left arm.

When first examined by Friedreich (being then 24, and having been ill nine years), he could still walk with help. He walked with a tottering, uncertain gait like that of a drunkard, with peculiar tossing movements of the legs, particularly well seen on going upstairs.

He had difficulty in standing, even with open eyes, and could only do so with tottering and balancing movements of the body. Delicate movements of the hands were made with much uncertainty and irregularity. All the sensory functions were unimpaired. The speech was thick and stammering. There was slight curvature of the dorsal spine.

He was re-examined three years later. The incoordination had increased, but except that he had become subject to vertigo, and had lost power of erection and emission, there was no additional symptom. Thirteen years later he came to Friedreich again. Now he could no longer stand, owing to difficulty in balancing himself, and even when sitting, his head, if unsupported, nodded and swayed. But still there was no true paralysis, and no affection of sensation. His speech was barely intelligible. A slight lateral nystagmus, only evident on fixing, had made its appearance. He died, a year later, of typhoid, at the age of 38, the duration of the spinal disease being twenty-three years.

Let us proceed to examine the clinical features of the disease more in detail.

The more striking feature is its tendency to attack many members of one family. With regard to ordinary tabes, most authorities agree that it does not run in families, so that this constitutes a first clinical point of difference. Not that Friedreich's disease is usually hereditary, in the strict sense of the term (though it may be so), but that it attacks many members of the same generation. Table I. shows this in every instance, with the possible exception of Brousse's case, who does not mention whether the patient had any brothers or sisters. Very remarkable in this respect are Rutimeyer's observations on the Blattner family; where in the same generation, from four separate branches of the family, eight members were affected. Yet that the disease may be transmissible is shown in this same instance, for it appears probable that a male ancestor five generations back was ataxic. Again, in Brousse's case the mother of the patient was ataxic; and in Carré's (besides the brothers, sisters, and maternal cousin) the mother, maternal uncles and aunts, and grandmother. So that successive as well as simultaneous outbreaks are possible, though not the rule. Apart from ataxy, proclivity to other forms of disease has been observed in those families; on the side of the nervous system, to chorea, paralysis, hysteria, mental affections, intemperance; on the side of general disease, to phthisis.

Friedreich's opinion, based on his own nine cases, was that females were much more prone to the disease than males, but subsequent observations show that this is not the case.

Secondly, it attacks in early life. Ordinary tabes is a disease of middle age or of later life. Doubtless we have now means of recognising it at an earlier period than formerly we could; but still its onset is less early than that of the hereditary form. Friedreich stated that the latter coincided with commencing or developing puberty, the dates in his cases varying from æt. 13 to æt. 18; but other authors have observed it to begin still earlier. Thus, in three of Rutimeyer's cases it began about the age of 4 (one child indeed had never been able to walk straight); and in two of Hammond's cases at a similarly early age. Table II. shows that for members of the same family there is usually a curious coincidence as to the dates of onset.

The first system is almost invariably ataxy of the lower limbs. This statement will hold, we believe, in spite of certain recent criticisms. Hammond, commenting on Friedreich's cases, says, "the first symptom observed was weakness of the lower extremities, and this gradually extended so as to involve the upper extremities. Here was, therefore, a true paralysis." But we must remember, that as early symptoms generally come through the report of the patient or his friends, any impediment to walking (whether paralytic or ataxic) would probably figure as weakness of the legs. If we look to the result of subsequent medical observation, we shall find many carefully observed cases in which there was ataxia but no paralysis. It is true that absolute paraplegia is recorded in some cases, and paresis of the lower limbs in others, but this was not till the disease had existed for years.

The absence of sensory symptoms is a point of great interest. In typical tabes, as is well known, the pains are usually a prominent symptom, especially in the early stages, and are rarely absent throughout. Further, sensory disturbances, such as numbness, anæsthesia, delay in the transmission of painful impressions, frequently accompany the incoordination of movement. But in the majority of hereditary cases pain is conspicuous by its absence, at least till the disease is inveterate. (There are a few exceptions, to which we shall return.) Again, though diminution of sensibility has been often noted, it is usually, for the earlier periods, in quite an insignificant degree, and in many cases every abnormality of sensation has been thoroughly excluded. We have, therefore, in this disease an illustration of ataxy pure and simple, apart from motor or sensory paralysis.

Harmony in muscular contraction is needed, says Friedreich, as much for the maintenance of a position as for movement. The lack of it during movement produces the staggering gait, the awkwardness of the hands, &c., i.e. locomotor ataxy; the same defect in the maintenance of position he calls "static ataxy." It is illustrated in this disease by the swaying of the head and neck when unsupported, by the wavering motions sometimes seen when the arms are held out, and is a later phenomenon than is locomotor ataxy. The well-known "Romberg's symptom," i.e. inability to stand with the feet together and the eyes closed, is ascribed by Rutimeyer to static ataxy in the muscles of the trunk. This is not always present. The disease generally spreads upwards with rapidity. In many cases the arms have been attacked very soon after the legs, or at least within a year or two. But this is not invariable; four, six, eight, or more years may elapse; indeed, in one of Dreschfeld's cases the arms had not been attacked after fourteen years.

The affection of speech may manifest itself either simultaneously with the affection of the arms or not till many years afterwards. A verbal description of peculiarities of speech is more difficult even than description of gait; but this much we may say, the defect is a defect of articulation, not of word-memory; neither on the other hand is there loss of motor power in the organs of speech, though tremor of the tongue has been noticed. From a mere drawl, stammer, undue confluence, or undue separation of syllables, the affection may advance till speech becomes wholly unintelligible.

Nystagmus marks clinically the highest level to which the disease extends. It is bilateral, usually transverse, and is observed only while the patient fixes or follows an object, It is a late phenomenon, and therefore its absence need not tell against the diagnosis.

We have now enumerated the most striking features of the disease, viz. ataxy, first of the legs, then of the trunk and arms, imperfection of speech, nystagmus. But there are others of importance. First, the absence of knee-phenomenon. This has been found absent in every case examined for it (the older cases of course were not). In Friedreich's last case it was found absent within a year after the disease had declared itself, and we might expect from analogy to find it absent still earlier. In the healthy children of an ataxic family Rutimeyer has found the knee-phenomenon normal. Secondly, curvature of the spinal column. In most nervous diseases (disregarding, of course, those secondary to disease of the vertebræ) the spinal column is unaffected. Pseudo-hypertrophic muscular paralysis may be quoted as an exception. And indeed, as in that case, so in this, the cause is essentially the same, viz. the lack of efficient muscular support. A combination of lateral and angular curvature is frequent; but lateral curvature alone is noted five times as against angular curvature once. This we should expect, assuming the cause to be laxity of the muscular ligaments. Curvature of the spine may be a very early symptom; in Carpenter's second case, and in one of Friedreich's cases, it manifested itself two years before the unsteadiness of gait. Its occurrence may be sometimes due to mere coincidence, but this can scarcely be so in every case, considering how frequently it is noted.

There remain sundry other symptoms, in a certain sense accessory, and appertaining mostly to an advanced period of the disease. Such are true paralysis, atrophy, and contracture of muscles, and, on the sensory side, anæsthesia or pains. Perhaps we may add occasional visceral disturbances. Rutimeyer has noted an early form of contracture, viz. that the great toes become turned up towards the dorsum of the foot; the mother of one family had herself observed this in her children, and put it down as a "malum signum." Such symptoms probably denote that tracts of the cord other than the posterior columns are involved. Interest therefore attaches to the date of their appearance, since it may help us to understand the manner in which the disease spreads within the cord.

It is worth while to recapitulate the clinical relations borne by this disease to the ordinary form of tabes. The phenomena of ataxy beginning in the lower limbs and spreading upwards, surely, if at a variable rate, coupled with the loss of tendon reactions, constitute the common basis of the two affections. The prognosis quoad recovery appears equally hopeless in each. But they exhibit differences. For, firstly, the multiform nervous phenomena which may usher in typical tabes—such as lightning pains, anomalies of micturition, transient paralysis (especially in the oculo-motor district) loss of pupillary reflex; or more rarely optic atrophy, visceral crises, trophic disturbances in joints, bones, and elsewhere—all these are absent in the hereditary form; where uncomplicated ataxia is the initial and for a time the only symptom. Particularly remarkable is the absence of pain, and indeed, at the outset, of any sensory or motor paralysis. Secondly, in the hereditary form the spread of the affection to the arms is more rapid; and the disturbance of speech and nystagmus are peculiar to it. If with Friedreich we regard these also as ataxic, then we may say that the ataxia spreads more rapidly and at the same time spreads further in this than in the typical form. We have only to add the differences in family predisposition and in the age at onset (already sufficiently noticed) to complete the contrast.

Now seeing how marked the contrast is, it is not surprising that some authors should have denied the existence of any relationship between the two diseases. Thus Hammond maintains that Friedreich's cases are not examples of locomotor ataxy at all, but of disease in the medulla or cerebellum extending secondarily to the cord. An answer to this assertion is to be found in the morbid anatomy. For not only Friedreich's post-mortems (which cannot, we think, be shelved simply on the score of being old) but also the recent researches of Schultze and Brousse establish the fact that the disease is not cerebral, but spinal, and involves the posterior columns. The same answer must be made to the French authors who regard it as a variety of disseminated sclerosis. The affection of speech and the nystagmus indeed lend clinical support to that view, but, post-mortem, the sclerosis (at least as regards the posterior columns) has been found to be systematic, not disseminated. The views expressed by Erb appear to us better to represent the facts. This author, while recognising fully the varieties exhibited by tabes, thinks that most cases may be grouped under one of two types, either the classical type or that described by Friedreich. This he regards as the only natural subdivision. To borrow botanical language, the natural order tabes contains two species, to which most individuals may be referred. The propriety of this classification is justified, rather than otherwise, by the occurrence of forms transitional between the two. Such forms are well illustrated by Carré's case (Table II., No. 21), in which there was well-marked heredity and affection of speech, yet the disease began with pains and numbness in the feet and legs, and diplopia was observed. Again, Dreschfeld's three cases (25–27), were it not for the fact that five members of the same family were attacked, resemble the classical type; for there were pains from the onset, and no affection of speech is mentioned. In both Carré's and Dreschfeld's cases the onset was rather late for that of the hereditary disease [æt. 20–23]. Again, early pains were noted in three of the Süss family (Nos. 3 to 5); and in Power's case (No. 34) there appear to have been vomitings at an early period. This appears to be the place to notice the observations of Seeligmüller. These relate to two brothers, whose parents were first cousins, and whose mother and maternal aunt were highly neurotic. The one brother had ataxy of the legs, nystagmus, and fibrillar twitching of the tongue, though his speech was normal. He was subject to migraine, dreamy states, and inverted sexual passions. The other brother was ataxic in the legs, had commencing nystagmus, and his speech was peculiar. He also was subject to dreaminess and forgetfulness. Friedreich refused to acknowledge these as cases of hereditary ataxy, doubtless on account of the mental symptoms, and because the knee-phenomenon was present. But they are worth mentioning while on the subject of aberrant forms, especially when we remember the connection between ordinary tabes and general paralysis of the insane.

The morbid anatomy of this disease rests upon six cases (Friedreich, Schultze, Brousse), in which the symptoms were well-marked during life, and careful examinations were made post-mortem, and upon one other case (Kahler and Pick) in which important anatomical data were obtained, though the clinical history was somewhat imperfect. Of these we shall give an abstract, as some parts of the pathology are still matter of discussion.

I. Andreas Lotsch (Table I., Case 1) died from typhoid when the ataxy was of twenty-one years' standing. Besides the ataxy and the affection of speech, there had been true paralysis of the adductors of the thighs, with some general feebleness of movement.

Post-mortem, the following lesions were found by Friedreich. Beneath the *dura mater*, in the lumbar regions of the cord only, there was fluid, and at a corresponding level some softening of the cord.

The *pia mater* upon the posterior aspect of the cord was milky and thickened in its whole length, and there were soft filamentous adhesions between it and the dura. The *posterior columns* were grey, translucent, and firm (this change being most marked, and appearing to have commenced, just above the lumbar swelling, and terminating just above the bifurcation of the posterior columns at the calamus); the rest of the interior of the cord was normal. Microscopically, there was found atrophy of the posterior nerve-roots, with slight changes in the nerve-trunks of the sciatics and hypoglossals.

The microscopic changes in the interior of this, and of all the other cords, appear to have shown the ordinary characters of sclerosis; there was an overgrowth of fine fibrillar tissue at the expense of the nerve-fibres, which were found in various stages of degeneration. The so-called corpora amylacea were found in abundance, especially in those parts where the sclerosis appeared to be commencing.

II. Justinie Süss (Table II., Case 3) died of typhoid in the fifteenth or sixteenth year of her disease. During life, in addition to the cardinal symptoms of ataxia, difficulty of speech and nystagmus, there had been noted curvature of the spine, occasional pains, cramps in the lower limbs, some tactile anæsthesia of the lower limbs and some general feebleness of movement.

The post-mortem (Friedreich) gave the following results. There were changes in the *dura and pia mater* closely resembling those in the last case, together with pigmentation of the cervical portion of the pia. The cord was flattened and somewhat softened, rather above the lumbar swelling. The *posterior columns* were sclerosed from the lumbar to the cervical swelling throughout their whole sectional area, above the cervical swelling the sclerosis affected only a thin zone next their periphery. In addition to this, the *lateral columns* were sclerosed, between the lumbar and cervical swellings, but to a less degree than the posterior columns, and only where just adjacent to them. There was, as in the last case, a slight extension of the posterior sclerosis to the medulla, and also atrophy of the posterior nerve-roots.

A further examination of this cord, made after many years by Schulze, showed that the grey matter also was sclerosed, chiefly in the dorsal region, and the central canal was stuffed with small round cells. The cells of Clarke's columns were diminished in number.

III. Salome Süss (Table II., Case 4) died of typhoid when her ataxia was of twelve or fourteen years standing. Here also during life there had been feebleness of movement, and at an early period shooting-pains, cramps, and spinal curvature.

Post-mortem, Friedreich found *meningeal changes* like those in the last two cases; also thickening of the ependyma of the 4th ventricle, and in the cord the following lesions; sclerosis of the *posterior columns* in their whole length, highly advanced in the part between the cervical and lumbar swellings, in which part the posterior fissure was obliterated, the surface of the posterior columns sunken, and the cord generally flattened. Microscopically, the *lateral columns* were found to be affected in the part where they abutted on the posterior columns. There was some atrophy of the posterior nerve-roots.

There were interesting changes in the grey matter, as follows: reaching from the lumbar swelling as far as halfway to the cervical swelling, were two canals with fluid contents, longitudinal in direction, and symmetrically placed one at each junction of the posterior and anterior cornua of grey matter, mostly within the grey matter, but just trenching on the lateral columns. These were prolonged upwards to the cervical region as foci of jelly-like material. There were less definite appearances of the same kind in the lumbar region.

Basing his remarks on these three post-mortems, Friedreich maintains[1] that the essential lesion consists in a chronic inflammation of the posterior columns of the cord, beginning in the lumbar region and extending upwards and downwards. But the affection of the posterior columns he was inclined to regard as secondary to the meningitis which he had found in all three cases.

The latter opinion he discarded in view of his next case.

IV. Friedrich Süss (Table II., Case 6) died of typhoid, having been ataxic some twenty or thirty years. There had been in his case no accessory symptoms, if we except vertigo and loss of sexual power.

The sac of the dura mater contained a good deal of fluid; the arachnoid was thickened and milky; the pia was thickened, and in the cervical region pigmented. The whole cord was thinner than normal, but the posterior columns specially small. An elaborate description of the minute changes, with plates, is given by Schultze.[2]

The *posterior columns* were sclerosed throughout (in the cervical regions the columns of Goll were the most deeply affected); this lesion extended upwards to the level of the calamus.

The posterior parts of the *lateral columns* were sclerosed in the cervical, dorsal, and lumbar regions (the intensity of the process, to judge from the plates, diminishing from above downwards). There was a thin zone of less degenerated tissue between the sclerosed portions of the posterior and lateral columns.

Except in the lumbar region, the affection of the postero-lateral columns reached quite to the periphery of the cord (sclerosis of direct cerebellar tract?); and in the cervical region there was a zone of degeneration extending round the periphery of the lateral and anterior columns to the anterior fissure.

Further the *R. anterior column* in the neighbourhood of the fissure was deeply sclerosed; this lesion reached upwards a little above the decussation of the pyramids, and had disappeared in the dorsal region of the cord (sclerosis of R. direct pyramidal tract?).

The grey matter of the cord was somewhat diminished in volume; the cells of Clarke's columns were remarkably atrophied.

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[1] Virchow's Archiv, Bd. 26, pp. 453, 454.

[2] Virchow's Archiv, vol. lxx.

The posterior nerve-roots were thinned; the peripheral nerves and spinal ganglia were normal.

The medulla oblongata was small; it showed no distinct sclerosis, but there was slight increase of connective tissue and numerous corpora amylacea in the restiform bodies.

These changes are read by Friedreich as follows: assuming an insufficient development of the cord and medulla (as evidenced by their size), upon this became grafted the posterior sclerosis, which he considered in this instance too intense to be secondary to the meningitis. Upon the posterior sclerosis followed the meningeal changes, which were the medium through which the disease was conveyed to other tracts of the cord than the posterior columns. But this explanation of the spread of the disease is contested by later writers, notably by Kahler and Pick, whose case we give next.

V. Case recorded by Kahler and Pick, in the 'Archiv für Psychiatrie,' &c., Bd. viii. p. 251.

This was a woman, age 23, whose sister was said to be affected in a similar manner. Her illness began when about 16 years old, with painful swellings of the ankles, then progressive weakness of the legs. In six years' time the arms became weak and the speech was affected. When she came under observation the muscular weakness was so preponderant that it was impossible to say whether there was ataxia or not. The tendon reflexes, however, were absent. There were no pains and no contractures. The speech was slow, the syllables separated and brought out with effort. She died of phthisis. The authors found, post-mortem, some adhesion of the pia and dura mater in the cervical and dorsal regions, but quite insufficient to account for the changes within the cord. The cord was smaller than normal, and the posterior nerve-roots thinned.

The *posterior columns* were degenerated throughout the whole length of the cord, except a thin zone next to the grey matter. The *postero-lateral* columns were also sclerosed right up to the periphery (crossed pyramidal tract and direct cerebellar tract), but here also a zone next the grey matter was spared. The *R. anterior column* in the neighbourhood of the fissure (R. direct pyramidal tract) was degenerated down to the dorsal region; the L. was not degenerated, but to make up for this, the degeneration in the crossed pyramidal tract on the R. side (of the cord) extended over a larger area than on the L. side. [This the authors point out would be the result, supposing there were (as is known to be sometimes the case) complete decussation at the pyramids of the L. pyramidal tract, with partial decussation of the R.] The columns of Clarke were atrophied.

The gist of this case, which the authors assimilate to those of Friedreich, though the clinical data are confessedly scanty, is to show that the sclerosis does not spread from one tract of the cord to another by simple contiguity (since zones of healthy tissue intervened), nor yet, as Friedreich supposed, by the medium of a meningitis; but that there is a distinct and independent degeneration of various tracts—of the posterior columns proceeding upwards, of the pyramidal tracts (crossed and uncrossed) proceeding downwards, of the direct cerebellar tracts, and with them of the columns of Clarke. They assert, therefore, that Friedreich's disease is not a simple posterior sclerosis, but a "combined systematic degeneration of the cord."

To this view it would appear Schultze gives his adherence,[1] though *à propos* of the case which follows, he criticises it adversely.[2]

VI. Charlotte Lotsch (Table II., Case 2) died finally of nephritis, having been ataxic for more than thirty years. In the later stages there had been paralysis of the lower limbs, with contracture of some muscles and atrophy of others; also cramps and pains.

Post-mortem, Schultze found *meningeal changes* much resembling those already described. The cord was somewhat small. There was sclerosis of the *posterior columns*, and of the posterior parts of the *lateral columns*. As in Case 4 (Fr. Süss), the periphery of the lateral columns was diseased, but the zone of degeneration did not reach so far forwards.

The *anterior column of each side* was sclerosed from the decussation of the pyramids downwards, the degenerated tract taking the form of a comma directed from behind forwards. The columns of Clarke and the posterior nerve-roots were degenerated.

The sacro-lumbales muscles were atrophied.

VII. The last case is that published by Brousse (Table II., No. 22).

This patient, a woman æt. 24, who was the subject of phthisis, died of subarachnoid (cerebral) hæmorrhage, having been ataxic for eight years.

The cerebrum was in other respects normal, the cerebellum also. The pons varolii was small. There was in this case no meningitis.

Minute examination of the cord showed the lesions to be most intense at the lumbar swelling. These lesions were as follows:—The *posterior columns* were sclerosed in the whole length of the cord; at the lumbar swelling this sclerosis extended to the periphery of the cord; in the cervical region it did not quite reach the periphery; it was most intense in the neighbourhood of the central canal and along the edge of the posterior cornua.

The *lateral columns* were sclerosed chiefly at the junction of the anterior and posterior cornua, the lesion reached the periphery of the cord in the lumbar region only.

The *anterior columns* were sclerosed principally in the neighbourhood of the anterior cornua; in the cervical region the lesion extended to the periphery of the cord (on the L. side only).

The sclerosis of the lateral and anterior columns was well marked in the cervical region, less marked in the dorsal, and most extensive in the lumbar, where the whole area of these columns was affected.

Further, there was well-marked inflammation of the *central canal*, which was full of small deeply-staining cells. Upon this Brousse lays stress, as he considers that by this means the disease spread from the posterior columns to the other parts of the cord.

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[1] Neurologische Centralblatt, vol. ii. p. 290. Report of a speech by him.

[2] Virchow's Archiv, Bd. 79, p. 136.

In the medulla oblongata there was a patch of sclerosis occupying the posterior pyramids and reaching to the level of the hypoglossal nucleus.

Brousse holds that the sclerosis of the posterior columns is primitive and systematic; while the sclerosis in other parts is secondary to it, and is diffuse, i.e. not limited to, nor propagated along, any particular tracts of fibres.

We believe that the above abstract represents with tolerable accuracy the facts already ascertained about the morbid anatomy of this disease; we will conclude by recapitulating them.

In several cases the cord and the medulla were small in size; and this is certainly a fact of interest when we consider the hereditary character of the disease. For a cord congenitally imperfect may be the more prone to degeneration, and to degenerate at an early age.

Next from the clinical history, as well as from the fact that advanced posterior spinal sclerosis was found in every post-mortem, we may assert that this at least is an essential lesion. The changes in the medulla are slight and apparently secondary to the spinal; so in Friedreich's opinion were those which he found in the nerve-trunks; while in the cerebrum and cerebellum no trace of chronic disease has been found. We therefore cannot adopt Hammond's suggestion that the lesion is primarily situated in the medulla or cerebellum.

Thirdly, since the sclerosis of the posterior columns involves their whole length, and appears to spread upwards much as in ordinary tabes, this we must class as a systematic tract lesion and not as part of a disseminated sclerosis.

But the relation of the disease in other parts of the cord to the posterior sclerosis is as yet obscure. Does such disease of other parts always exist? if so, has it spread by simple contiguity, or by a meningitis, or by the medium of an inflammation attacking the central canal? Or have we here an instance of systematic posterior sclerosis combined with disseminated sclerosis of other parts; or, on the other hand, of systematic sclerosis of many spinal tracts at the same time?

To answer these questions we need further examinations, and especially examinations at early stages of the disease.

TABLES.

TABLE I.—To show the Genealogical Relations of most of the Published Cases.[1]

Friedreich. 1. Lotsch Family:— Father intemperate, died of dropsy. Mother died of sudden hemiplegia.	There were	5 children, of whom 2, viz. one male and one female, *were diseased.* Table II., 1 and 2. 3 were healthy.
2. Süss Family:— Father intemperate, died of phthisis. Mother living, healthy, but of a low grade of intelligence.	There were	5 children, of whom 4 *were diseased,* one male and three females. Table II., 3 to 6. 1 was healthy.
3. Schulz Family:— Father and mother living, healthy country people. No history of disease in ancestry.	There were	11 children, of whom 5 died young. 3 were healthy. 3 (all females) *were diseased.* Table II., 7 to 9.
4. Brousse's Case:— Father's mother subject to hæmoptysis.	Father emphysematous, died, æt. 70, of cerebral softening. Mother died, æt. 38, was said to be ataxic, like her daughter.	*The patient,* a female, died of phthisis. Table II., 22. No note whether she had brothers or sisters.
5. Carré, as quoted by Brousse:— *Grandmother* (maternal?) *was ataxic.*	*Mother ataxic.* *Mother's brothers and sisters, 8 in number, all ataxic.*	There were 12 children, of whom 5 were not affected, 7 *were ataxic,* viz. the patient (female). Table II., 21. 4 brothers, 2 sisters. *One cousin (male) was also ataxic.*

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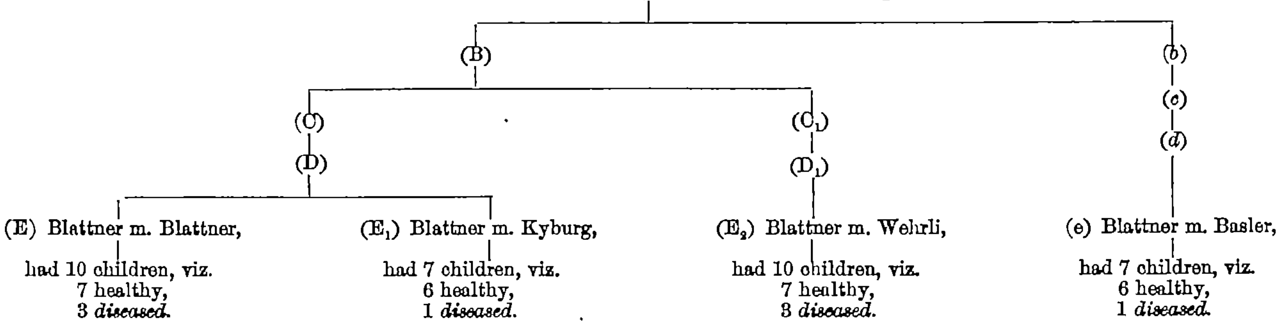
[1] We omit from these tables the case by Kahler and Pick, which is chiefly of anatomical interest; those by Seeligmüller, which Friedreich did not admit to be cases of hereditary ataxy; those by Kellogg and by Schmid, the original account of which we have not been able to obtain; and one or two cases quoted by Hammond.

TABLE I.—To show the Genealogical Relations of most of the Published Cases (*continued*).

Rutimeyer.

6. Blattner Family —Four families, in each of which there were cases of the disease, were descended in the male line from a common great-great-great-grandfather (who was nicknamed the "Stumbler") as follows;—

Blattner (A), nicknamed "Stulpi," *presumably ataxic* ?



(B)

(C) — (D)

(C₁) — (D₁)

(*b*) — (*c*) — (*d*)

(E) Blattner m. Blattner, had 10 children, viz.
7 healthy,
3 *diseased.*

(E₁) Blattner m. Kyburg, had 7 children, viz.
6 healthy,
1 *diseased.*

(E₂) Blattner m. Wehrli, had 10 children, viz.
7 healthy,
3 *diseased.*

(e) Blattner m. Basler, had 7 children, viz.
6 healthy,
1 *diseased.*

See Table II., 10–17. Of the 8 ataxics, 6 were males, 2 females.

In the case of the Blattner-Basler branch there were nervous complaints (chorea, poliomyelitis) on the mother's side.

Quincke and Rutimeyer.

7. Kern Family :—

Father's family and father quite healthy.
Mother's father intemperate, and during last part of his life mentally affected.

Mother suffered from "general nervousness."

There were 10 children, of whom
7 were healthy,
3 *were diseased*, 1 male, 2 females.
Table II., 18–20.

Carpenter.

8. Father healthy.
Father's brother died of general paralysis.
Father's cousin peculiar, hysterical ?
Mother (and her family) healthy.

There were 8 children, of whom
5 were healthy,
1 healthy at the time of Dr. Carpenter's observations.
2 *were diseased*, females. Table II., 23, 24.

9.	*Gowers.* SHAW FAMILY:— Father's mother was paralysed in her tongue.	Father died of Bright's disease. Father's brother ditto. Another brother died insane. Mother died late in life of abdominal tumour; in early life had two attacks of chorea.	There were 9 children, of whom 1 died, æt. 10 years, 3 (males) were healthy, 5 *were diseased*, viz. 4 males and 1 female. Table II., 29–34.
10.	*Dreschfeld.* Father, æt. 72, living and healthy. Mother died, æt. 54, of bronchitis.		There were 15 children, of whom 10 were not affected (3, however, died quite young). Two of these 10 were married, and had healthy children. 5 *were diseased*, 3 males and 2 females (viz. Table II., Cases 25–27, and two who had other patients died previously).
11.	*Hammond.* No disease of the kind known in previous generations.		*Two brothers were diseased.* Table II., 35, 36.
12.	No neurosis known in the family.		Ditto. ditto. Table II., 37, 38.
13.	Ditto. ditto.		Ditto. ditto. Table II., 39, 40.
14.	*Wickham Legg and D'Arcy Power.* In grandparents and other ancestry there was no nervous disease known.	Father healthy, and has 3 brothers living, healthy, 1 brother dead of small-pox. 2 sisters living, healthy, 1 sister dead of phthisis. Mother healthy, and all her brothers and sisters healthy.	There were 10 children, of whom 7 were healthy, 3 *diseased*, viz. the patient, a male, the eldest. Table II., 34. One brother (dead) and one sister, who, according to the patient, suffered like himself.

TABLE II.—ABSTRA

No. in present Table.	——	Age and symptoms when first attacked.		Duration of disease when seen by Author.	*Ataxic symptoms.* Gait, lower and upper limbs; head and neck. Romberg's symptom.	*Motor system.* α, True paralysis; β, nutrition γ, electro-irritability of musc δ, Contractures; ε, tremor η, choreic movements.
1	Andreas Lotsch (Friedreich, Case 1).	18	Difficulty in walking.	19 years. [watched for 2 years, when death from typhoid.]	Cannot stand or sit upright. Head oscillates. Cannot lift cup to mouth, or take hold of an object easily.	Can move lower limbs, with effort; adductor thighs paralysed. Musc power of upper limbs mal. Muscles act to tricity.
2	Charlotte Lotsch (Friedreich, Case 2).	18	Weakness, first of left then of right leg.	13 years, 31 years [and for 2 years till death from nephritis].	Cannot stand without support. Cannot pick up things, nor thread a needle. Later, oscillations of the head.	Movements of lower limbs formed with effort; fu paralysis, with contrac of some muscles, atroph others. Upper limbs, paralysis.
3	Justinia Süss (Friedreich, Case 3).	16	Weakness and wandering pains, in legs.	15 years.	Gait ataxic: needs support, even for standing. Fine movements of hands difficult. Slight oscillations of head and neck.	Simple movements perfor with effort, but electrotractility of muscles mal. Had bad involun flexions of lower limbs, cramp.
4	Salome Süss (Friedreich, Case 4).	14	Curvature of spine, weakness and pain in legs 2 to 3 years later.	14 or 12 years.	Cannot stand or sit upright. Cannot button clothes nor thread needle.	Simple movements perfor feebly and with effort. I tro-contractility of mu normal.
5	Lisette Süss (Friedreich, Case 5).	15	Weakness of legs, then of arms. "Gnawing" pain in bones.	17 years.	Bedridden for years. Movements of hands ataxic. Head and trunk oscillate.	Simple movements req much effort.
6	Friedrich Süss (Friedreich, Case 6).	15	Weakness of left leg and arm, then of right.	9 years. 12 years. 25 years.	Gait that of a drunken man. Totters as he stands. Worse in dark or with eyes shut. Fine movements of hands difficult. Oscillations of head late in the disease.	Muscular power and ele contractility normal.

IDUAL CASES.

ensory system. nsibility to touch, , and temperature; cular sense; electro- uscular sensibility; ooting-pains, girdle pains, cramp, &c.	Reflex actions; tendon reactions; skin-reflexes.	Spinal column.	Organs of speech; speech; movements of tongue and lips.	Organs of vision (and other special senses).	Visceral vaso motor secretory functions.	Cerebral functions.
mal, except for a ninution of elec- -sensibility in e muscles. No ins.		Normal.	Speech stammering and difficult to understand. Fine tremor of tongue.	Normal.		Normal.
nal. Cramps and ins in lower limbs, vards the later riod of the dis- se.	Tendon-reactions absent. Skin-reflexes present.	Kypho-scoliosis becoming worse.	Speech thick and stammering, finally unintelligible.	Nystagmus late in the disease.	Polyuria, salivation, &c. Some bladder difficulty (later).	Occasional vertigo.
ile sensibility di- nished in lower nbs and abdomen. ectro - muscular nsibility dimin- ed. *Occasional ins*, sometimes ndering, some- nes shooting, from rliest period.		Kypho-scoliosis said to have appeared early.	Speech stammering and often unintelligible.	Nystagmus.		
tro-muscular sen- ility diminished. casional shooting *ins* from early od, and cramps.		Kypho-scoliosis.	Speech heavy and stammering; intelligible.	Slight nystagmus.	Palpitation (systolic murmur). Cough and dyspnœa.	Frontal headache.
ns in legs fre- ent: gnawing ins an early mptom; later eurulgic" pains; ally, painful mps in various rts.		Kypho-scoliosis.	Speech affected.	Normal.	Palpitation and dyspnœa.	Had had hysterical attacks. Recently vertigo.
mal in every way.			Speech heavy and stammering, finally unintelligible.	Slight nystagmus late in the disease.	Loss of sexual power.	Vertigo.

TABLE II.—Abstra

No. in present Table.	——	Age and symptoms when first attacked.		Duration of disease when seen by Author.	*Ataxic symptoms.* Gait, lower and upper limbs; head and neck. Romberg's symptom.	*Motor system.* α, True paralysis; β, nut γ, electro-irritability of δ, Contractures; ε, tremors η, choreic movements.
7	Louise Schulz (Case 7, Friedreich).	13	Stumbling gait, clumsiness of hands (headache 2 years before).	5 years. 9 years. [Watched for 3 years each time.]	Well-marked ataxia of movement, both in upper and lower limbs; not increased by closure of eyes. (Later, inability to stand or sit without irregular movements of head and trunk.)	No loss of power. Mus natural. Later, sl twitching of finger, h &c.
8	Katharina Schulz (Case 8, Friedreich).	13	Helplessness of legs, then of arms (headache previously).	3 years. [Watched for 2 years more.]	Similar to last case (but symptoms rather less marked, and not progressing while under observation).	Normal.
9	Marie Schulz (Case 9, Friedreich).	13	Upper and lower limbs affected at nearly the same time.	1 year. 4 years.	As in the two last cases. (Later, staggered during standing.)	Normal.
10	Rudolf Blattner (Case 1, Rutimeyer).	7	Uncertainty in walking and standing (especially in the dark).	10 years.	Walks with feet apart, staggering. Sways as he stands. Worse with eyes shut. Difficulty in buttoning clothes, &c. Slight oscillation of head and trunk as he sits.	No paralysis. Muscles ulnar side of right fore slightly wasted. Some regular movements w he holds out arms. Sli flexion of some of fingers, and dorsal flex of great toes.
11	Gottlieb Blattner (Case 2, Rutimeyer).	7	Weakness and uncertainty in walking.	27 years.	Ataxic stage had passed into that of paralysis when patient came under observation.	Lower limbs almost paral Muscles atrophied. Up limbs paretic atroph Contracture of some fing flexion of knees, dou equino-varus, dorsal flex of great toes.
12	Marie Blattner (Case 3, Rutimeyer).	6	As in the two last cases.	23 years.	Unable to walk. Writing, buttoning clothes, &c., very awkwardly performed.	Lower limbs paretic, wasted. Contractures fingers and hands, sli equino-varus, dorsal flex of toes.
13	Rudolf Blattner, β. (Case 4, Rutimeyer).	7	Fatigue and uncertainty in walking, especially in the dark.	8 years.	Walks with feet apart, staggering. Swaying of trunk as he stands. Worse with eyes shut. Complicated movements of hands awkwardly performed.	Nutrition normal, except t left side of face is less veloped than right. dorsal flexion of great

DUAL CASES—*continued.*

ensory system. sibility to touch, and temperature; ular sense; electro- cular sensibility; ting-pains, girdle ins, cramp, &c.	Reflex actions; tendon reactions; skin-reflexes.	Spinal column.	Organs of speech; speech; movements of tongue and lips.	Organs of vision (and other special senses).	Visceral vaso-motor secretory functions.	Cerebral functions.
e normal. [Later, ght diminution sensibility to ich, and to elec- city in the es ?]	Tendon reactions absent. Skin-reflexes normal.	Normal.	Words uttered slowly and with abrupt pauses. Movements of tongue and palate normal.	Normal.	Intercurrent albuminuria (? nephritis).	Headache (migraine ?) preceded onset of disease.
ıal.			Speech affected.	Normal.		Do.
aal.	Tendon-reactions absent. Skin-reflexes normal.		Speech affected soon after limbs.	Nystagmus when last observed.	Normal.	Normal.
it diminution of ctile sense in ver limbs (?), ierwise normal.	Tendon-reactions absent. Skin-reflexes normal.	Slight scoliosis.	Speech drawling, nasal, with frequent change of pitch. Slight tremor of tongue.	Nystagmus developed when under observation.	Slight systolic apex murmur. Subject to hiccough.	Normal.
le sense di- nished in both per and lower ıbs. Conduction pain delayed.	Tendon-reactions absent. Skin-reflexes normal.	Marked kyphosis and scoliosis.	Speech slow, stammering, almost unintelligible.	Nystagmus. Deafness.	Frequent sweatings and salivations. Hiccough on drinking.	Normal ?
ile sense di- nished in upper d still more in ver limbs.	Tendon-reactions absent. Skin-reflexes normal.	Marked kyphosis and scoliosis.	Speech drawling, but intelligible.	Nystagmus. Sight and hearing said to have been affected.	Cyanosis and œdema of legs and feet.	Normal.
ıal. (Particu- ly sensitive to ctric current.)	Tendon-reactions absent. Skin-reflexes normal.	Slight kyphosis and scoliosis.	Speech drawling, somewhat nasal.	Nystagmus developed while under observation.		

TABLE II.—ABSTR

No. in present Table.	——	Age and symptoms when first attacked.		Duration of disease when seen by Author.	*Ataxic symptoms.* Gait, lower and upper limbs; head and neck. Romberg's symptom.	*Motor system.* α, True paralysis; β, nutritio γ, electro-irritability o δ, Contractures; ε, tremo η, choreic movements.
14	Karl Blattner (Case 5, Rutimeyer).		Never learnt to walk straight. Gait more uncertain last few years.	Boy (age not stated).	Walks in zig-zags. Difficulty in complicated movements. "Writes over the lines." Gait worse with eyes shut. Jerking movements of head.	Twitchings of fingers a isolated muscles wh holds out arms.
15	Maria Magdalene Blattner (Case 6, Rutimeyer).	4 or 5	Arms and legs simultaneously affected. Speech not till 8 or 10 years later.	15 years.	Can only walk with support; throws legs about. Complicated movements of hands and arms uncertainly performed. Symptoms not worse when eyes are shut.	Paresis of lower limbs. I flexion of great toes.
16	Jacob Blattner (Case 7, Rutimeyer).		As in previous case. Disease followed chorea.	Not seen by author.		Paralysed like Gott No. 11.
17	Fritz Blattner (Case 8, Rutimeyer).	4	Uncertainty in walking.	Not seen by author.		
18	Heinrich Kern (Case 9, Rutimeyer and Quincke.)	6 or 7	Ditto.	6 years and 13 years.	Waddling gait. Head and trunk sway as he stands. Worse with eyes shut. Ataxia of arms less than of legs, not worse with eyes shut.	At time of second observ almost complete paraly lower limbs. Wastin muscles, esp. below k Contractures at k double equino-varus flexion of great toes.
19	Hedwig Kern (Case 10, Rutimeyer and Quincke.)	7 or 8	Ditto.	8 or 9 years.	Jactitation of legs in walking. Needs support. Head and trunk sway as she stands. Not worse with eyes shut. Movements of arms very uncertain.	No paralysis. Slight wave of arms when held out. sal flexion of great toes ing last year).
20	Bertha Kern (Case 11, Rutimeyer).	7	Ditto.	6 years.	Much as in last case, though rather less marked.	No paralysis. Slight d flexion of great toes.

UAL CASES—*continued.*

nsory system. dibility to touch, and temperature; lar sense; electro- lar sensibility; ing-pains, girdle ns, cramp, &c.	Reflex actions; tendon reactions; skin-reflexes.	Spinal column.	Organs of speech; speech; movements of tongue and lips.	Organs of vision (and other special senses).	Visceral vaso-motor secretory functions.	Cerebral functions.
al. (Tactile se diminished highs ?)	Tendon-reactions absent. Skin-reflexes lively.	Slight kyphosis and scoliosis.	Speech slow, drawling, and nasal.	Nystagmus.		
e sense dimin- ed in legs. Oc- ional formica- .	Tendon-reactions absent. Skin-reflexes normal.	Slight kyphosis.	Speech drawling, clumsy. Fibrillary tremor of tongue.	Nystagmus.	Cyanosis and œdema of feet.	
			Affected.			Chorea at first. Fits, in one of which he died, æt. 18.
			Not affected (?) but died at 9 of measles.			
e sense mark- y diminished in ver limbs, htly so in up- (At first only tle formication eet.)	Tendon-reactions absent. Skin-reflexes normal.	At first normal; later marked kyphosis and scoliosis.	Speech indistinct, tongue cannot be held still. Afterwards speech worse, only slight tremor of tongue.	Nystagmus (at second observation).	Cyanosis of feet, slight œdema.	Vertigo among the early symptoms.
e sense slightly inished (?) in er limbs.	Tendon-reactions absent. Skin-reflexes normal.	Scoliosis (during last year).	Somewhat drawling. No tremor, &c., of tongue.	Slight nystagmus.		
e sense slightly inished (?) in upper and limbs.	Tendon-reactions absent. Skin-reflexes normal.		Speech not affected.	Slight nystagmus.	Some coldness and cyanosis of legs and feet. Obscure affection of knee at age of 5.	

TABLE II.—Abstr

No. in present Table.	—	Age and symptoms when first attacked.		Duration of disease when seen by Author.	*Ataxic symptoms.* Gait, lower and upper limbs; head and neck. Romberg's symptom.	*Motor system.* α, True paralysis; β, nutritio γ, electro-irritability c[illegible]es δ, Contractures; ε, tremor η, choreic movements.
21	Carré's case, female, as quoted by Brousse.	22	Pains in feet and legs; then numbness and difficulty in walking.	6 years.	Peculiar gait; short, jerky steps. Disorderly movements of head and eyes as she walks. Not worse with eyes shut. Fine movements of hands disorderly.	No paralysis.
22	Brousse's case, female.	24	Weakness and incoordination of legs in walking.	8 years.	Can walk only with support: throws feet forward and outward. Difficulty in picking up pins, &c. Worse with eyes shut; cannot stand. Occasional tremor of head.	No paralysis. Swaying m ments of arms when stret out.
23	Female (Case 1, Carpenter).	12	Unsteady gait. Spinal curvature.	17 years (at time of report).	Gait uncertain, as if intoxicated: foot brought down forcibly, heel first. Some clumsiness of hands. Worse with eyes shut.	No paralysis. Electro-tractility diminished low knees. Tendenc talipes equinus.
24	Female (Case 2, Carpenter).	12	Spinal curvature. Irregular gait 2 years later.	15 years (at time of report).	Similar symptoms (but less marked) to those in last case. Sight less necessary to her. Head and shoulders shaky.	Electro-contractility (in below normal.
25	Male (Case 1, Dreschfeld).	20	Weakness in walking; esp. in dark. Shooting-pains.	22 years.	Fine movements of hands difficult. Ataxy of legs merging into paralysis.	Simple movements of (when recumbent) slow jerky. Paresis of Grasp weak. Mus flabby.
26	Male (Case 2, Dreschfeld).	23	Weakness of knees. Pain in lower limbs.	9 years.	Jactitation of feet as he walks. No ataxia of upper limbs. (At first, difficulty of walking in dark.)	Atrophy of muscles of ha forearms and legs. G weak. Diminution of tro-contractility. (Mu of face slightly atrophi
27	Female[1] (Case 3, Dreschfeld).	20	Sharp pains. Heaviness in walking.	14 years.	Ataxic gait: cannot walk with eyes shut. No ataxia of arms.	Muscles of thighs so somewhat flabby, b paresis.

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[1] Two other members of the same family

OUAL CASES—*continued.*

nsory system. dbility to touch, nd temperature; ar sense; electro- sensibility; pains, girdle ns, cramp, &c.	Reflex actions; tendon reactions; skin-reflexes.	Spinal column.	Organs of speech; speech; movements of tongue and lips.	Organs of vision (and other special senses.)	Visceral vaso-motor secretory functions.	Cerebral functions.
in early part disease, but sen- y functions ved quite nor- l.			Speech stammering, as if tongue were fixed for a moment. Tremor of tongue when put out.	Diplopia and supposed imperfection of sight.		
al.	Tendon-reactions absent.		Speech hesitating and sputtering. Movements of tongue normal.			Convulsions in infancy. Intelligence low; irritable, &c. Died in fit (subarachnoid hæmorrhage).
t and occasion- numbness in s; tactile sen- ility slightly ered? Electro- scular sensi- ty normal.	No plantar reflex.	Lateral curvature.	"Precision in speech," as if effort to co-ordinate.	Normal.	Skin of legs easily frozen by ether-spray.	Normal.
al.		Lateral curvature.	Speech hesitating as if there was something under tongue.	Normal.	Ditto.	Normal.
e sensibility h diminished lower limbs. ns at onset of ase.			Normal.	Normal.		
o-muscular sibility dimin- d in legs. Oc- onal numbness, no loss of tac- sense. Pains nset.			No abnormality mentioned.	Vision impaired at first (since normal).	Loss of sexual power.	
sensibility hat dimin- in legs (pains nset).			Normal.			

been affected in a similar fashion.

TABLE II.—ABSTRA

No. in present Table.	—	Age and symptoms when first attacked.		Duration of disease when seen by Author.	*Ataxic symptoms.* Gait, lower and upper limbs; head and neck. Romberg's symptom.	*Motor system.* α, True paralysis; β, nutrition γ, electro-irritability of musc δ, Contractures; ε, tremors η, choreic movements.
29	Male (Case 1, Gowers).	21	Unsteady walk.	18 years.	Gait not remarkably ataxic; legs lifted too high if he takes long steps.	Some weakness of legs.
30	Female (Case 2, Gowers).	18	Difficulty in walking.	11 years.	Difficulty in standing out of proportion to muscular weakness. Ataxy of arms. Head steady.	Ditto.
31	Male (Case 3, Gowers).	19	Ditto (?).	4 years.	Takes short steps and sways about. Head unsteady. Can only stand with feet apart. Arms ataxic: can barely write.	No paresis.
32	Male (Case 4, Gowers).	20 (?)	Slight unsteadiness.	2 or 3 years (?).	Difficulty in standing with eyes shut. Arms natural.	
33	Male (Case 5, Gowers).	?		?	Unsteadiness in turning. Cannot stand with eyes shut. Handwriting unsteady.	No paresis.
34	Power's case. Male.	15	Loss of power in legs. Vomitings.	7 years.	Cannot even sit upright. Head nods and falls forward. Buttons clothes with difficulty; cannot do it with eyes shut.	Wasting of legs (and thigh farado-contractility mal. Extension of performed with efforts. I jump at night. (Was of each 5th met.ca phalangeal joint.)
35	Male (Case 1, Hammond).	4	Fell when walking.	5 years. 15 years.	At first observation could not stand; at second, could not sit up.	All four limbs said to "paralysed," but legs be moved as he lies dov
36	Male (Case 2, Hammond). Brother of last.	10½	Weakness of legs.	6 months. 10½ years.	"Gait staggering, but not like locomotor ataxy." Not worse with eyes shut. At date of second observation could not walk without help. Arms affected.	

UAL CASES—*continued.*

ory system. bility to touch, mperature; ense; electro- ar sensibility; ng-pains, girdle s, cramp, &c.	Reflex actions; tendon reactions; skin-reflexes.	Spinal column.	Organs of speech; speech; movements of tongue and lips.	Organs of vision (and other special senses).	Visceral vaso-motor secretory functions.	Cerebral functions.
ility to pain eas[illegible] other- [illegible] abnor- ity.	Tendon-reactions absent. Skin-reflexes mostly active.		Syllables run together, sometimes unduly separated.			
al.	Tendon-reactions absent.		Speech markedly syllabic.			
l.	Tendon-reactions absent. Skin-reflexes mostly active.		Articulation confluent.			
e sensibility aired in legs. erwise no ab- mality.	Tendon-reactions absent. Skin-reflexes lessened.		Tendency to confluent articulation.			
Ditto.	Tendon-reactions absent. Skin-reflexes mostly present.		Articulation sometimes syllabic, sometimes confluent.			
of shooting- in back. t impairment actile sensibil- n legs. Numb ing in arms and t.	Tendon-reactions absent. Skin-reflexes normal.	Lateral curvature (lumbar).	Speech jerky, sometimes slow, sometimes rapid, slurring. Difficulty in mastication, and swallowing.	Optic discs rather white, otherwise normal.	Vomitings (at first). Some incontinence of urine.	Weak intellect. Had attacks of giddiness with loss of consciousness and falling.
al.	Skin-reflexes normal.		Speech barely intelligible.	Normal.		Normal.
			Speech indistinct at date of second observation.			

TABLE II.—Abst

No. in present Table.	—	Age and symptoms when first attacked.		Duration of disease when seen by Author.	*Ataxic symptoms.* Gait, lower and upper limbs; head and neck. Romberg's symptom.	*Motor system.* α, True paralysis; β, nutrit γ, electro-irritability of m δ, Contractures; ε, tremo η, choreic movements
37	Male (Case 3. Hammond).	6	Stumbling. Fatigue in walking.	4 years.	Cannot walk without help. Gait said to be that of paralysis, not ataxy. Not worse with eyes shut. Head falls forward as he sits.	"Upper limbs weak, he cannot feed hi Nutrition normal. (lar spasms before de
38	Male (Case 4, Hammond).	8	Weakness in legs.	Course of disease like his brother's (last case).		
39	Male (Case 5, Hammond).	3	Weakness first of right then of left leg.	13 years. 27 years.	Gait staggering: sways a little with eyes shut. Later cannot stand with eyes shut.	Movements of legs ma effort. Hands weak, tro-contractility Later, involuntary ings of hands.
40	Male (Case 6, Hammond).	7	Weakness of legs.	19 years.	Symptoms as in last case (his brother), only less pronounced.	

AL CASES—*continued.*

system. lity to touch, temperature; sense; electro- ar sensibility; pains, girdle cramp, &c.	Reflex actions; tendon re-actions; skin-reflexes.	Spinal column.	Organs of speech; speech; movements of tongue and lips.	Organs of vision (and other special senses).	Visceral vaso-motor secretory functions.	Cerebral functions.
l. (Severe efore death.)			Speech barely intelligible. Difficulty in swallowing.			Frequent vertigo. Mind ill-developed.
ns till just death.						
ok of head neck (on l observa-	Tendon-reactions absent.		Difficulty in pronouncing words: often forgets them.	Nystagmus (on second observation). Sight bad at night.		Vertigo.
	Tendon-reactions absent.			Deaf left ear, from measles.		

Reviews and Notices of Books.

Recherches sur le Système Nerveux des Poissons.—Par EMILE BAUDELOT, professeur à la Faculté des Sciences de Nancy. Ouvrage précédé d'un avertissement par M. EMILE BLANCHARD, et accompagné de 10 planches gravées. Paris; in folio; 1883. G. Masson.

THIS very remarkable and important work is a posthumous one, and has been published by the care of Madame E. Baudelot, the widow of the distinguished scientist, and M. E. Blanchard, the well-known naturalist, and president of the Academy of Sciences in Paris. Although the author died before having investigated the subject as thoroughly as he intended to do, this book may be considered as presenting a fair sketch of what it would have become if the author had lived. Baudelot had written a good deal on fishes generally, and had studied their nervous system very accurately. The subject had been rather overlooked: little was known concerning that question. In the present review we must leave the greater part of the book untouched, and confine ourselves to the chapter dealing with physiological questions. Concerning the functions of the central nervous system, M. Baudelot has made some interesting experiments. As Desmoulins and Magendie have already shown, the loss of the cerebral hemispheres does not interfere with the power of motion, nor with any special function. Fishes move about as easily and as unconcernedly as if nothing had been done to them.

The loss of the superior part of the optic lobes does not affect fishes any more than that of the cerebral hemispheres in regard to movement, but the eyesight seems impaired, the fish remains quiet, as if it were asleep. If the inferior part of the optic lobes is destroyed or injured in some manner, a great change in the movements is noticed. The fish turns round and round on the long axis of its body; it falls on the side opposite to the injured one, and goes on spinning in the same manner during many days, sometimes a fortnight. This rolling motion may be executed ten, twenty, even a hundred or more times per minute. When it stops, the fish lies on the side opposite to the injury; the body is also more or less bent up on the same side (*mouvement de manège*). Baudelot believes that the rolling motion (*mouvement de rotation autour de l'axe*) may be ascribed to a painful feeling of contracture.

If the medulla oblongata is injured, there is also a rolling motion, but quite a different one: the motion is contrary to that of the preceding experiment: the fish lies on the injured side. The injuries of the cerebellum do not seem to affect motion nor sensation, unless the medulla oblongata is also injured.

Generally speaking, injuries of the cerebral hemispheres do not affect motion, nor sensation, nor intellect in fishes, while they do affect voluntary motion and intellect in mammalia; the injuries of the cerebellum impair motor co-ordination in mammalia, but do no harm to fishes. Injuries of the superior part of the optic lobes affect fishes, as those of the corpora quadrigemina affect mammalia; injuries of the inferior part of the same lobes affect fishes, as those of the optic thalami and cerebral and cerebellar peduncles affect mammalia.

Although the physiological part of Baudelot's work is not a very long one, it is of much importance in the study of cerebral physiology generally.

Of the remainder of the book we shall only say that it is full of interesting information: it was not to be expected that there could be in the cerebral form of the different sorts of fishes the variety that M. Baudelot shows to exist.

The engravings are excellent: they add a great deal to the value of this book, and help us greatly to understand the general morphology of the brain, as explained by the much regretted and distinguished zoologist. Mme. Baudelot deserves much praise for not having allowed her husband's valuable researches to be lost; M. E. Blanchard and G. Masson are also to be complimented for their generous attachment to science which induces them, especially Mons. G. Masson, to spend much time and assume much trouble in pursuits that are generally known to be more costly than profitable.

HENRY DE VARIGNY.

Clinical Lectures on Mental Diseases. By T. S. CLOUSTON, M.D. &c. London: J. and A Churchill; 1883. Crown 8vo. pp. 631. Illustrated.

MR. G. T. ROMANES has lately published two books. In one of these he narrates a vast number of instances of intelligent conduct on the part of animals; in the other he investigates deeply the constitution of mind and the workings of the nervous system, and deduces certain broad general principles which he elucidates and illustrates by means of the facts contained in his first volume. The first book is a storehouse of data, a granary of facts, a museum of instances, a treasury of records, a warehouse of raw material: the second is the manufactured article, it is the history which the records disjointedly narrate, it gives the laws to which the instances apply, it draws the conclusions from the data, it displays the utilisation of the facts. The first book is purely narrative, the second mainly argumentative. The danger in the second book is of fallacy; the danger in the first is of "anecdotage."

Dr. Clouston's book fulfils, in a different province, nearly the same function as Mr. Romanes' first work on Animal Intelligence. It is one long record of accurate and laborious observations. From cover to cover the book is positively bursting with facts. It presents to the student of alienism an almost embarrassing profusion of data. The number of cases which are recorded in full is considerable; and the skill with which the salient points of each case have been chosen for presentation, and the graphic terseness with which they are described, will be fully appreciated by those only who have attempted similar tasks with less success. The records of cases are far from being the only materials placed at our disposal in this interesting volume. Repeatedly at frequent intervals the results of Dr. Clouston's ripe experience are put before us in the form of empirical generalisations, which, representing multitudinous observations, are equivalent to large groups of facts.

Nor is this all, for in addition to the excellent therapeutics with which the book is saturated, we meet with many shrewd remarks, and occasionally with a flash of true philosophic insight. A few samples will whet the reader's appetite for more. "There is no use," says Dr. Clouston, "in dunning a patient to 'rouse yourself,' to 'throw off your dulness,' to 'drop those fancies,' for in many cases it would be just as wise to tell a hemiplegic to 'move that leg.'" "We think of melancholia chiefly from the patient's subjective point of view, taking his affective change and his conscious mental pain chiefly into consideration; while we think of mania more from our own objective point of view, and picture the patient's talkativeness, his restlessness, and his manifest changes of personality and habits." "The ravings of a maniacal patient are often well worthy of study. . . . There is no such thing as real incoherence. The words and ideas always cohere by some bond or other." The homicidal feeling "is undoubtedly the human instinct of slaughter and destruction." It "is analogous to the physiological instinct of breaking things in children." It is no doubt a case of "throwing back," of atavism. It is a reappearance in excess of the pugnacious instincts of our savage ancestors. "Why," asks Dr. Clouston, speaking of the ill-effects of high-pressure education upon growing girls, "why should we spoil a good mother by making an ordinary grammarian?"

Like every other writer upon insanity, Dr. Clouston proposes his own classification; of which it may be said that nothing would be more easy than to pull it to pieces and display its inconsistency, nothing more difficult than to construct a better. In the chapter on Epilepsy, Dr. Clouston exhibits a curious but by no means unique misapprehension of Dr. Hughlings-Jackson's views on this subject, the doctrines attributed to Dr. Jackson being such as he would shudder to hear of.

While acknowledging the admirable manner in which Dr.

Clouston has accomplished the task to which he committed himself when he undertook to write a book of Clinical Lectures on Insanity, it is right to point out the limitation of that task. Of the science of Insanity, as of every other science, there are two aspects; and of these Dr. Clouston limits himself to one. Nothing could be clearer than the way in which he presents that aspect of the subject with which alone he undertakes to deal; but it is necessary to remember that this is only one of two aspects, and the view that he gives us is unavoidably imperfect. He has given us knowledge; but knowledge is not science until it is organised; and the organisation is wanting. To render the work complete we want a book that shall treat the subject from the opposite point of view, and be to Dr. Clouston's present work what Mr. Romanes' second book is to his first. We have the facts; we now want the principles. We have the data; we now want the conclusions. We have the instances; we now want an enunciation of the laws to which they conform. We want an account of the workings of the normal mind and of the normal nervous system; an explanation of the ways in which it is possible for them to become disordered; a description of the results of such disorder, illustrated by the instances which Dr. Clouston's book affords in such abundance. Facts accumulate upon us, until we stagger under the burden of them, and are fain to tumble them down here and there in disorderly heaps. Our most urgent need is of an architect who will provide some plan upon which we can build them up into a regular and useful structure.

CHARLES MERCIER, M.B.

A Practical Introduction to Medical Electricity. By A. DE WATTEVILLE, M.A., M.D., B.Sc., &c. Physician in charge of the Electrical Department at St. Mary's Hospital, London, &c. Second Edition. London: H. K. Lewis, 1884. 8vo. pp. 220; with 89 woodcuts, and five plates of the motor points.

THE employment of electricity in practical medicine has encountered vicissitudes so great and of such variety, as make it a matter of some wonder that anything satisfactory survives the trying ordeal to which it has been exposed. Beyond the well-meaning, but self-deceived enthusiasts who have published exaggerated accounts of its utility, and the impudent quacks who have traded on the mystery of its force, there have, however, happily been cautious and persevering observers whose communications have been always tending to bring order out of chaos; and we may hope that a time is within sight when the place of the electric current in the practice of our profession will be more or less definitely fixed.

In his earlier edition (which was noticed three years ago in these pages) the author rendered excellent service to the cause of progress by giving us, what had previously to a great extent been wanting in English works upon electro-therapeutics, solid and accurate information upon the theory of electricity, so far as concerned us as physicians. The present condition of his work is so enlarged, and for the most part re-written, as to constitute a new book. In it the author has still further endeavoured to popularise such elementary facts in electrophysics as will go far, if mastered by the reader, to preserve him from misconception, and keep him upon the right track for a really scientific observation of such phenomena as present themselves to him when using electrical currents in the practice of medicine. Dr. de Watteville still insists upon measurements of current strength as the essential condition of a rational application of electricity to medicine, and advocates the constant employment of the galvanometer. We would direct especial attention to the part of his book in which he deals with this question. It contains much valuable information and judicious advice. So also, as regards the question of electrical distribution or diffusion, we think he makes a decided point in suggesting the adoption of Professor Erb's set of standard electrodes, so as to fix, once for all, the meaning of the terms "small," "large," "moderate sized," so frequently used. The relative position of the electrodes upon the body in various applications of currents is considered very fully, and the diagrams borrowed from Erb, which are introduced to illustrate the subject, are clear and eminently descriptive. Nearly 100 pages are devoted to these and other questions in electrophysics, together with descriptions of the apparatus most serviceable in medical practice.

Chapter II. deals with electro-physiology. A novel feature is here introduced. The instruction is given in the form of experiments which the reader may perform upon himself. These are numbered, and arranged for reference, one to another. This is an excellent method, by which a thoroughly practical idea of the subject may be obtained, and a variety of erroneous notions dispelled.

We have been disappointed that Dr. de Watteville has not preserved the German notation for marking qualitative changes in the excitability of nerve and muscle. As we have before suggested, if the word "shut," be used instead of "close," as regards the circuit, and the letter Z (Zuckung = contraction) be accepted as an arbitrary sign implying contraction (and there is something in the form of the letter suggestive of it), records made by English and most foreign observers will be mutually intelligible. Faraday introduced the terms, "anode" and "kathode," which have been adopted by the Germans. In this country instead of the letters K S Z for "kathodic shutting contraction," the letters C C C, "cathode closure contraction," have just got to be pretty generally used, when Dr. de Watteville again disturbs the harmony by employing K instead of C, so that now we have K C C. Surely amongst all these diversities we are in danger of losing sight of the Q. E. D.! The adoption of the German notation would have saved all this inconvenience, and it is, we think, to be desired that English-speaking observers may even yet be led to see the advantage of it.

The subject of electro-diagnosis is considered in Chapter III., and the numerous difficulties and sources of fallacy in investigations are pointed out in a very graphic manner. Chapter IV. is devoted to electro-therapeutics. In it the author shows that he is anxious that the reader should clearly understand the rationale of the methods of applying the current to the human body.

The work is well illustrated by numerous engravings, amongst which the plates showing the motor points strike us as exceptionally clear. There is no doubt that this book, based as it is upon all the recent advances in electrical science, must take a most important place amongst works in English upon the subject of electricity as applied to medicine, and we congratulate the author on his achievement of a difficult task.

T. Buzzard, M.D.

Clinical Lectures on Giddiness. By Thomas Grainger Stewart, M.D., Professor of the Practice of Physic in the University of Edinburgh, &c. 8vo. pp. 43. Edinburgh, 1884.

This pamphlet consists of three clinical lectures devoted to the discussion of the symptom giddiness. The subject is treated in a comprehensive manner, and at the same time in a concise and intelligible form. The author, after a short description of what is understood by the term, proceeds to investigate its relation to disorders of the sensory and motor functions. It is explained that equilibrium is not a condition born with the individual, but is acquired as the result of education, and is thereby subservient to the tactile, ocular, muscular, articular, visceral, and labyrinthine impressions. It is pointed out that the semi-circular canals are the chief sensory organs presiding over this function, and for its proper performance there must be integrity of these as well as of the nervous centres, and the conducting nerve fibres, change in any of which may lead to abnormal modifications, which among other symptoms are manifested by vertigo. In health all the peripheral and central conditions harmonise to preserve normal equilibrium; where there is any break in this arrangement, or where there is "a contradictoriness of sensory impressions," giddiness is the result. This may be due to peculiarities in external conditions, and more especially through the medium of the sight, as evidenced by the peculiar sensations experienced when we look from a height or at a distorted object, such as the Tower of Pisa, or in waltzing, swinging, or being on board ship. Again, giddiness may be due to abnormalities of the sensory structures themselves, as seen in strabismus, diplopia, nystagmus, &c. The most important form of vertigo is that resulting from causes situated in the semi-circular canals, and which is known as Menière's disease. Various varieties of this have been described, as the external, middle, or internal ear is affected. It is pointed out that giddiness may arise from diseases of the spinal cord as in locomotor ataxy, multiple sclerosis, &c., and may be explained partly by the defective action of the muscles of the eye, and partly from changes in other sensory impressions. Vertigo may arise from a variety of intra-cranial abnormalities, and amongst those especially referred to are those localised in the cerebellum, crura cerebelli, pons, cerebral peduncles, and corpora quadrigemina. Besides these the symptom may be the result of various diseases of the brain, either organic or functional, such as tumours, congestion, anæmia, injuries, epilepsy, megrim, disease of the arteries, effects of galvanism, &c. A variety of morbid conditions of the general system may be followed by the same derangement as in nervous debility, dyspepsia, toxic infection, fevers, constitutional cachexias, and so on. The explanation of the occurrence of giddiness as a result of these is somewhat doubtful, but is probably to be explained by their effects on the blood, and its abnormal influence on the nerve centres.

Contribution à l'étude et au diagnostic des formes frustes de la Maladie de Basedow. Par M. le Dr. Pierre Marie. Paris, 1884.

The author of this little book does not claim that it is a complete monograph on the subject of Basedow's disease. It professes only to discuss a certain number of those marked symptoms which are not usually described in association with the malady, or which have been considered as purely accessory. These, on the contrary, M. Marie believes to be intimately connected with the affection, constituting peculiar and characteristic features, sufficient to enable us to detect the disorder, even when the special symptoms of goitre and exophthalmus are wanting. The general conclusions arrived at are—

1. Tremor is a frequent symptom of Basedow's disease, and is probably almost always present.
2. This tremor has special characters which permit it to be distinguished from others.
3. The presence of goitre or exophthalmus, either together or singly, are not essential elements of the disease.
4. When this tremor accompanies palpitation of the heart, without notable augmentation of the temperature, Basedow's disease may be suspected. The diagnosis will be confirmed by the presence of one or more of the following symptoms:—Local or general sweating, boulimia, insomnia, cough without expectoration, or abnormal physical signs in the chest.

A. Hughes Bennett, M.D.

Abstracts of British and Foreign Journals.

Ferrier and Yeo on the Effects of Lesions of the Cerebral Hemispheres. (*Proceedings Royal Society*, xxxvi. 229.)—The following is an abstract of a paper on this subject recently presented to the Royal Society, and which contains a detailed account of a series of observations on the effects of localised lesions of the brain of monkeys, made partly by Drs. Ferrier and Yeo, and partly by Dr. Ferrier alone.

The operations were performed under anæsthetics, and according to the rules and methods of antiseptic surgery.

The experiments are illustrated by photographs, showing the position and extent of the lesions and general condition of the brains, together with sun-prints and microphotographs of sections of the brain and spinal cord, showing the structures primarily involved or secondarily degenerated.

As the paper is mainly an anatomical description of the lesions delineated and record of the effects produced, it scarcely admits of condensation in the form of an abstract. The lesions are classified according to their position in the occipito-angular, temporal, Rolandic, frontal, and hippocampal regions respectively.

Among the more important results arrived at are the following:—

Lesions of the occipito-angular region (occipital lobes and angular gyri) cause affections of vision, without affections of the other sensory faculties or motor powers.

The only lesion which causes complete and permanent loss of vision in both eyes is total destruction of the occipital lobes and angular gyri on both sides.

Complete extirpation of both angular gyri causes for a time total blindness, succeeded by lasting visual defect in both eyes.

Unilateral destruction of the cortex of the angular gyrus causes temporary abolition or impairment of vision in the opposite eye—not of a hemiopic character.

Deep incisions may be made in both occipital lobes at the same time, or the greater portion of one or both occipital lobes at the same time may be removed without any appreciable impairment of vision.

Destruction of the occipital lobe and angular gyrus on one side causes temporary amblyopia of the opposite eye and homonymous hemianopia of both eyes, towards the side opposite the lesion.

As in none of the cases recorded, either of partial unilateral or bilateral destruction of the occipito-angular region, were the amblyopic or hemianopic symptoms permanent, it is concluded that vision is possible with both eyes if only portions of the visual centres remain intact on both sides.

Destruction of the superior temporo-sphenoidal convolution on both sides causes complete and permanent loss of hearing, without other sensory or motor defect. Hearing is not impaired by lesions of any other part of the temporal lobe.

Destructive lesions of the Rolandic zone (convolutions bounding the fissure of Rolando) cause motor paralysis, without loss of sensation, limited (monoplegia) or general (hemiplegia), according to the position and extent of the lesion.

Lesions of this region are followed by descending degeneration of the pyramidal tracts of the spinal cord.

Lesions of the frontal region vary according as they affect the post-frontal (base of the frontal convolutions) or pre-frontal (anterior two-thirds of the frontal convolutions) region.

Lesions of the pre-frontal regions alone do not produce any discoverable physiological symptoms.

Lesions of the post-frontal regions cause temporary paralysis of the lateral movements of the head and eyes.

As the symptoms are only temporary so long as any portions of the frontal lobes remain intact, it is concluded that the post-frontal and pre-frontal regions have essentially the same physiological signification, and that portions only of the frontal lobes are sufficient for the exercise of their functions.

Lesions of the frontal lobes are followed by descending degeneration of the mesially situated tracts of the foot of the crus cerebri and corresponding fibres of the internal capsule, as seen in transverse sections. The ultimate destination of these tracts is uncertain. They cannot be traced in the anterior pyramids.

Lesions entirely destroying the hippocampal region (the hippocampus major and gyrus hippocampi) and neighbouring inferior temporo-sphenoidal region (without implication of the crus cerebri, basal ganglia, or internal capsule) cause complete anæsthesia—cutaneous, mucous, muscular—of the opposite side of the body, without motor paralysis.

The degree of impairment of tactile sensibility in those cases where it was not entirely abolished, is in proportion to the amount of destruction of the hippocampal region.

Lesions of the cornu ammonis alone, or gyrus hippocampi alone, do not cause permanent impairment of tactile sensibility. The duration of the effects of total destruction of the hippocampal region has not been determined.

No impairment of tactile sensibility has been observed in connexion with any of the other cerebral lesions described.

A. DE WATTEVILLE.

Abstract of an Essay upon the Limitations in Time of Conscious Sensations. Read before the Royal Society, Edinburgh, December 1883. By JOHN B. HAYCRAFT, M.B., F.R.S. (Edin.) &c., Professor of Physiology at Mason and Queen's Colleges, Birmingham.—If any sensory surface be stimulated for a given period, a sensation will be produced, which will not exactly correspond with this stimulation in point of time. The sensation will be produced a certain interval after the first movement of application of the stimulus, and will remain a certain time after the withdrawal of the stimulus. It follows from this, that two stimuli follow the one another sufficiently rapidly will give rise to a single sensation. The correspondence in time between the stimulus and sensation varies in the case of the different senses.

In experimenting with tactile impressions rapidly following one another, it was found that if the stimuli are at first applied slowly (1 to 40 per second), they give rise each to a distinct and separate feeling. Stimulate more rapidly, and they are fused, and a feeling or sensation is produced which is termed "roughness." If the impacts follow one another faster than 1400 per second, the sense of "roughness" disappears and a sensation of single impact is produced. It is to be remarked that the knowledge of the cause of the roughness is gained by experience only, and could not be learned by the tactile sense alone. The same sensation called "tickling" is produced, whether an impact stimulates a number of sensory areas one after the other, as in drawing a feather over the skin, or if the impacts follow one another on the same sensory area; for impacts of from 50 to 1400 per second, if applied to the same sensory area produce "roughness" if forcible enough, but when lightly applied, they produce "tickling."

Different areas of the skin vary in sensibility. In more sensitive parts localisation in space is more precise than in those parts less sensitive. The experiments of the author, however, show that in the least sensitive parts, *i.e.* skin of the back and chest, stimuli following one another rapidly are as easily distinguished in consciousness as when applied to the more sensitive parts, *i.e.* skin of lip and finger-tip. A study of thermal sensation shows that the correspondence in time between the stimulus and produced sensation varies with the thinness of the epidermis at that part. In no case is the correspondence very exact, an appreciable interval intervening between the application of a cold body and a sensation of cold. The difference in the exact limits of the feelings seen in the different sensations was then shown by the author to be due not to any difference in the centrally produced feelings, but to the differences seen in the translation of the external energy into nerve energy through the end organs. Thus a sensation of touch follows almost immediately the application of the stimulus, which directly affects the end organs; on the other hand, some time elapses before a cold body is felt; the withdrawal of heat from the end organs having to take place through a more or less thick layer of epidermis.

In the sensations of sound and sight, as well as in the tactile sensibility, when stimuli are repeated more and more rapidly, a period preceding complete fusion is reached when the sensation produced is disagreeable in its nature. In the case of sound, the irritation is produced by "beats;" the cause of "dissonance" in music is due to recurring stimuli not to be separated in consciousness, and yet not rapid enough to give rise to a simple sensation of sound. The flickering of a flame is also irritating in its nature, and tickling in the case of tactile sensibility may give rise to excruciating agony. We may compare, therefore, roughness or tickling—tactile sensations—to dissonance in music.

J. B. HAYCRAFT.

Adamkiewicz on Cerebral Pressure (*Sitzungsber. der Akad. d. Wissensch. zu Wien*, 1883).—The author has had the opportunity of observing a tumour of the cord which occupied the greater portion of the left lateral column between the 5th and 7th cervical roots, and compressed the anterior horn to a marked degree. No symptoms had been noted during life, however, which gave a clue to such a lesion. This case formed the starting-point of investigations by the author concerning cerebral pressure on the one hand, and the question of compressibility of nervous masses on the other. The first point only is discussed in the present paper. He concludes that a pathological increase in the pressure of the cerebro-spinal fluid (in other words in the "cerebral pressure") does not exist. This view is in complete opposition with the generally accepted theories on the influence of agents which diminish the intra-cranial space; but is supported by a series of experiments too considerable for us to adduce or criticise here.—The author describes the phenomena usually ascribed to cerebral pressure being due to cerebral irritation.

Sigmund Fuchs on the Histogenesis of the Human Cerebral Cortex. (*Ibid.* 1883).—The chief questions which the author has proposed to himself are: (1) At what period of the fœtal or extra-uterine life do medullated fibres (chiefly those which run parallel to the surface) make their first appearance in the cerebral cortex? (2) At what period are their number and calibre so far developed as to represent a system of fibres functionally equivalent with that in the adult?

He came to the following conclusions:—The new-born child possesses no trace of medullated fibres, neither in the central nor in the cortical portions. They are found in the former at the end of the first month only; in the cortex they appear at different times in the different layers. In the upper layer none are discovered before the fifth month; in the second layer, after the first year. The radiating bundles of the deeper layers appear in the second month; whilst the horizontal fibres of the third layer are found to be medullated in the eighth month.

During the seventh and eighth year the medullated fibres reach the disposition which they preserve throughout adult life.

The cortical medullated fibres, which in after-life are the thickest, are the first to make their appearance. We may assume that the chronological differences in the development of the medullated fibres correspond to their physiological interpretation. Pyramidal cells of the typical structure occur in the cortex at the time of birth. Deiter's connective tissue cells were clearly observed in the fifth month of child-life.

Zuckerkandl on the influence of the growth of Sutures and of the Cranial Shape in the direction of the Convolutions (*Medic. Jahrb.* 1883, Part IV.). Many observers have tried to prove that there are certain relations between the shape of the skull and the direction of the convolutions; in the doliohocephalic brain they thought it to be more longitudinal, in the brachycephalic more transverse. If this be the case, the usual direction must be departed from whenever external circumstances (such as intra-uterine pressure, premature synosteosis of the sutures, &c.) alters the normal development of the skull. The author closely examined six brains from skulls deformed by synosteosis; he found the altered shape of the brain persists after removal, and that the convolutions present the alterations just described. He also examined the brain of a child whose head had been subjected to abnormal pressure "in utero," on account of the deficient amount of amniotic fluid, and observed similar phenomena.

We may go still further and show that under normal circumstances the sutures exert an influence upon the convolutions. The frontal gyri have not everywhere the same shape, breadth, thickness and direction. Quite in front they are usually narrow, thrown into numerous folds, and connected with one another by transverse pieces. Posteriorly, when they pass into the ascending frontal they are wide, thick, much straighter. This dissimilarity is easily explained by the topographical relations between the convolutions and the coronal suture. As long as the skull grows rapidly, the convolutions will lengthen longitudinally wherever the skull expands in that direction; this is the case for the posterior part of the frontal gyri, which are situated in the region of the coronal suture. Anteriorly and inferiorly, however, where no suture allows of such a ready expansion, the growing convolutions must become more concentrated, closely pressed together and thrown into folds. Similar considerations obtain with reference to other parts of the cerebral surface.

V. Rogner, On the Variations in the Cerebral Fissures in the Hare, the Sheep, and the Pig (*Zeitschr. für Wissensch. Zoologie, vol.* 39).—Numerous variations are to be observed in the disposition of the cerebral sulci of these animals, even on the almost smooth brain of the hare. The author reaches the important conclusion, that these variations are not to be considered as mere anomalies, but as transitional forms between the type of sulci in one kind of animals, and that observed in other kinds.

H. OBERSTEINER, M.D.

BRAIN.

JULY, 1884.

Original Articles.

ON THE SENSORIAL LOCALISATIONS IN THE CORTEX CEREBRI.[1]

BY PROFESSOR LUIGI LUCIANI.

Director of the Physiological Laboratory at the Royal Institute for Superior Studies in Florence.

WHOEVER has made experiments on the sensorial centres knows how great and how many are the difficulties there are in proving the existence of disorders, total or partial, unilateral or bilateral, of the several senses. There is in this kind of research a constant danger of confusing objective facts with the individual appreciation of them. I therefore took pains to render more secure and rapid the diagnosis of sensorial alterations in animals whose brains have been operated upon, by making a comparative study of the value of the various methods employed, and finding new means better adapted to my object. I shall limit myself here to a sketch of the most general method of investigation applicable to the safe and rapid discovery of the chief forms of tactile, visual, auditory, and olfactory disturbances which present themselves in consequence of the extirpation of various cortical areas. This diagnostic method which, when employed methodically, in every case makes the results obtained perfectly comparable among themselves, consists in the observation and the definition of the behaviour of the animal under the various stimuli which act upon its several senses, during the time it is taking its food, with its eyes open or with its eyes bandaged, or again with one eye open and the other closed. One prepares for the animal some favourite article of food; for instance, boiled meat divided into small pieces, and when the hour for the meal has arrived, when the appetite of the animal is keenest, its favourite food is shown it and given it to taste; then its eyes are well closed by means of sticking-plaster. Now, especially if it be the first time, the animal will make with its leg a few efforts to remove it, but if a piece of meat is immediately thrown out to it it leaves the plaster alone, and occupies itself in a search for the food, sniffing vigorously with its nose close to the ground, and moving about with great rapidity. This is the most opportune condition for testing the tactile, auditory and olfactory senses: it is enough to touch the various points of the skin, on the limbs, head or trunk with a straw or a pellet of cotton wool fixed to the end of a small rod, that the animal should under normal tactile conditions suddenly react, quickly turning the head towards the part touched, whilst it goes on seizing the food; it remains passive, or almost so, however, if the points touched are the seat of the diminution of the general sensibility. This mode of testing the sense of touch gives such clear results as to leave nothing to be desired.

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[1] This paper contains an abstract, or preventive communication, of the chief results obtained by the author in a recent series of long and laborious experimental researches on the sensorial localisations in the brain of the dog and the monkey. The full memoir is to be published shortly in Italy. The present abstract has been translated by myself from Prof. Luciani's original Italian manuscript.—The reader will find abstracts of Luciani and Tamburini's previous researches in 'BRAIN,' Vol. I. p. 529; Vol. II. pp. 234 and 580; a criticism of Munk by Ferrier, Vol. I. p. 229; a paper by Ferrier on Amblyopia, Vol. III. p. 457; and an abstract of Ferrier's recent researches on Localisation in the present volume, p. 139.—A. DE WATTEVILLE.

By throwing pieces of meat at intervals upon the plate, sounds are produced more or less perceptible according to the size of the pieces and the force with which they are projected. The animal then has not only the sense of smell, but also that of hearing to divert its search. As soon as it has swallowed a piece of meat it stands motionless, pricking its ears in order to catch the direction of the sounds of other pieces falling, and if the hearing is perfect on both sides, it quickly turns either to the left or to the right, according as the food is thrown nearer to one ear than the other; whereas if one of the two sides (or both) is deficient, the animal behaves in such a way as to show the side on which audition is imperfect (or manifests the bilaterality of the lesion). This is the best means of discovering even slight and unilateral alterations of hearing; whilst the test with the diapason (made to vibrate in the proximity of one or other ear), calls, loud and sudden noises are of use for investigating more profound auditory troubles.

This method of testing by the mode of alimentation with closed eyes is also of use to appreciate, though less accurately, alterations in the olfactory sense. For this purpose, it is enough to eliminate the intervention of the sense of hearing, by first disposing on the ground pieces of meat at a certain distance from one another, and noticing the rapidity with which the animal finds them by olfaction only. Then, after obstructing one or the other nostril with a plug of cotton wool, and holding the animal in the arms, one gradually brings its nose towards a piece of meat; one can thus judge comparatively of the acuteness of the sense in the two nostrils by measuring the distance at which the meat lies, when the animal begins to sniff and stretch its nose. The rapidity with which it reacts to more or less powerful odours (ammonia, acetic acid, chloroform, turpentine, cloves, &c.) may fulfil the same purpose.

It is a pity that we cannot test with the same ease alterations in the sense of taste, which we have no other means of appreciating than the readiness with which the animal reacts to bitter substances (colocynth, quinine, digitaline, &c.) applied to the two sides of the tongue.

The careful examination of the visual sense is of the greatest importance, in order to secure a diagnosis of its disturbances, whether partial or total, bilateral or unilateral, such as hemiopia, amblyopia, psychical blindness, complete and absolute blindness. The best method here is still the test by alimentation, whilst one of the eyes is closed or both are open. Thus, for instance, after the left eye has been closed, small pieces of meat are thrown to the animal (avoiding the production of any sound) first in a line with its nose, or a little to the left, so that the images be thrown upon the external segment of the right retina; secondly, to the right, so that the images be thrown upon the internal half of the same retina. The animal turns at once to seize its prey if the section of the retina on which the image falls is normal, but shows that it does not perceive it, if that part of the retina be blind; or finally shows that it perceives, but directs itself towards the food with some hesitation, and not in the precise direction if the retina be amblyopic. It is, in general, difficult to distinguish between hemiopia and hemi-amblyopia; more difficult to distinguish between amblyopic phenomena and those of psychical blindness—an expression by which we intend to designate, with Munk, a peculiar condition in which the animal preserves the capacity of seeing, but has lost that of recognising objects. In order to succeed in this diagnosis it is necessary to place on the ground small pieces of meat along with pieces of sugar, or some other inodorous and non-alimentary substance of the same size. If the animal is simply amblyopic, it has only to approach the pieces of sugar in order to recognise them by mere sight without smelling or tasting them; but if it is psychically blind, if it has lost the memory of visual signs, it requires more than sight to recognise them, viz., smell and taste. This test gives sufficiently clear results in dogs, very evident ones in monkeys.

In certain cases, dogs operated on at certain points of the cortex show themselves completely blind to food placed in small pieces on the ground; in order to find them they employ smell and touch only. Still, we have not to do here with complete and absolute blindness (cortical, as Munk calls it), but perhaps with a psychical blindness of an extreme type with or without amblyopia. For if the animal be led into the open air in a garden where it has been accustomed to stay, one sees it proceed straight along the paths, successfully avoiding the contact of hedges, walls, and other fixed obstacles which it comes across; whilst on the other hand, it constantly strikes against any unusual obstacle, such as a board, placed across the path which the animal is following.

II. After this short description of the methods of investigation applicable to the organs of nerve, I shall now give an account of the most important results obtained by me. I shall speak first of the cortical centre of the sense of vision, which has been the principal object of my researches, because it is the one which lends itself to the most searching physiological analysis.

The first question which presents itself with reference to the visual centre or sphere refers to the localisation of the same in the dog and the monkey. My new experiments point to the necessary conclusion, that appreciable visual disturbances follow not only extirpation of the *occipital,* but also the *parietal, temporal,* and *frontal* lobes. According to some results, the complete or partial destruction of the *cornu ammonis* also produces marked disorders of vision. Analogous results induced Goltz to admit, that in dogs the visual centre is so diffused in the cortex, that there cannot be said to exist any localisation of it. But if we consider more closely the effects of decortications of the various lobes, it is found, that lesions of the *frontal* and *temporal* lobes give rise to transitory visual troubles which gradually diminish, and finally disappear altogether (or at least become inappreciable), after a few days or weeks. Similar lesions of the *occipital* and *parietal* lobes produce permanent results; a certain improvement takes place during the first few days after the operation, but they then persist unaltered for months, or during the whole time of the observation.

We have observed another fact which does not agree with the doctrine of Goltz. When the lesions in the frontal and temporal regions are of small extent, that is to say, comprise but a single segment, sufficiently circumscribed, of these lobes, they may give rise to no appreciable disorder of vision, whereas equally limited lesions in the occipito-parietal province constantly produce evident visual disturbances.

We shall not be able to interpret these facts, considered in their whole and in their particular, without appealing to the theory that the visual sphere—though chiefly concentrated in the occipito-parietal zone of the hemisphere—is not clearly circumscribed therein, but enters into relation with other centres, as it were "par engrenage," by means of direct anatomical prolongations.

The experimental results obtained on monkeys are in accordance with this mode of interpreting the facts which relate to the visual sphere of the dog. My new experiments confirm on the one hand the capital importance of the occipital lobes as centres of vision, as already demonstrated by Munk, whilst on the other they confirm the view, that these centres spread by irradiation into the parietal lobes, especially into the angular gyri, and their fold towards the parieto-occipital fissure, as was shown by Tamburini and myself, and by Ferrier and Yeo. My new researches prove in addition, that the isolated destruction of the angular gyri produce slight, and rather transitory, visual disturbances; and that the effects of destruction of the occipital lobes are aggravated then considerably, but not rendered permanent by a posterior decortication of the angular gyri.

In order that the analogy between the visual sphere of the dog and that of the monkey should be complete, one would have to demonstrate, that extensive decortication in the neighbourhood of the temporal lobe of the monkey gives rise to transitory but appreciable visual troubles. The facts we have elicited bear out the statement, that in the monkey also the visual centres present such connections with other centres, as are considered by us as the organic condition of the great psychological principle of the association of sensations and ideas.

The second question relative to the visual centres consists in the determination of the relations existing between the retinal field of each eye, and both their relative cortical centres. My recent experiments confirm the view that the fibres of the optic nerve of the dog undergo in the chiasma an incomplete decussation, as was seen by Tamburini and myself, and that the extirpation of one of the occipital lobes produces a bilateral homonymous hemiopia, as Munk was the first to prove clearly, though more extensive in the eye of the opposite side. From this fact, now recognised as constant, it is logically concluded, that each of the visual spheres has a direct relation with the more extensive internal segment of the retina on the opposite side (including about two-thirds of the retinal field), and with the less extensive external segment of the retina on the same side (including about one-third of the retinal field). But it does not follow from the fact that the direct and crossed fibres of the optic nerves correspond to two distinct segments of the retinal area, that they be distributed to distinct sections of the visual sphere, nor that each definite point of the retina have a fixed and constant relation with definite points of that sphere. I have obtained three different series of results which do not allow me to accept this conclusion.

1. Bilateral homonymous hemiopia results not only from complete extirpation of one occipital lobe, but also from extensive decortications of one parietal, and also of one temporal lobe.

2. Partial bilateral extirpations in the province of the occipital lobe (visual sphere of Munk) never produce distinct phenomena of partial blindness, but visual troubles, more or less accentuated, equally or almost equally diffused over the several segments of the two retinæ.

3. The hemiopic effects consecutive to extensive unilateral extirpations in the occipito-temporal and parietal region of the vortex, and the visual troubles diffused throughout the whole retinal field which occur after circumscribed bilateral extirpations in the same region, are not permanent but transitory phenomena.

These three groups of facts are, on the one hand, destructive of the well-known theory of Munk of the projection of the several segments of the retina upon definite segments of the visual sphere; on the other, they prove that though the crossed optic fibres arrive from the internal, the direct from the external retinal segments, they do not preserve their relative position throughout their long course, and in both cases enter promiscuously into relation with the ganglion elements of their respective segments of the visual sphere. Fig. A represents schematically our cortical visual centre or sphere: the dark points, which greatly predominate in the great occipito-parietal area of the left hemisphere, indicate the relations which the optic fibres coming from the internal segment of the right retina bear to the cortical centre of vision; the light points, which are more thinly scattered over the same region, indicate the relations which the optic fibres coming from the external segment of the left retina, bear to the same centre. It is easy to understand how, on this hypothesis, the three groups of facts first mentioned find a natural and adequate explanation.

The same theory applies to the visual centre of the monkey. The only well-demonstrated fact of Munk's (and which is confirmed by my recent researches) is the bilateral homonymous hemiopia which immediately follows the extirpation of an entire occipital lobe. But according to the doctrine of Munk this phenomena should be permanent, whilst my results show it to be temporary, and reproducible by repeated partial extirpations of the same lobe. This confirms the fact that the visual sphere of the monkey extends beyond the limits of the occipital lobe, and proves that each segment of the visual centre is related with the fibres of the crossed tract as much as with that of the direct fasciculus of the optic nerve—just as what we have shown to obtain in the dog.

The third problem to solve is that concerning the functional nature of the visual cortical centres. Are the visual *perceptions* only, or are also the simple visual *sensations*, localised therein? (taking these expressions in the definite sense assigned to them by the followers of Flourens). Or in other words, the hemiopic effects consequent on extirpation of the whole visual centre on one side, and the absolute blindness following their complete bilateral extirpation, are they definitive and permanent phenomena as Munk believes; or do they (as happens after incomplete uni- or bilateral extirpations) tend to become gradually attenuated, until they become reducible to simple psychical blindness?

My own researches, though still incomplete with reference to the determination of the definitive effects of total extirpation of our visual centres, are however more than sufficient to be opposed to the theory of cortical blindness, as much as to that of the absolute and permanent blindness which Munk believes to have observed in consequence of the complete bilateral extirpation of his visual sphere (circumscribed, as we know, to the two occipital lobes). The fact is that after the most extensive extirpation of the occipito-temporal area, which is far more extensive than the visual sphere of Munk, absolute blindness does not persist beyond a few days, and may even fail to supervene at all, if the extirpation is made by successive operations. The place of absolute blindness (when it occurs) is taken by a complete psychical blindness, through which the animal seems blind to any food placed on the ground before it, but seems still capable of avoiding fixed obstacles met on the way, and to follow the path in a straight line. Later on, the psychical blindness becomes incomplete, but yet permanent; the animal begins again to make use of sight in the search for its food, but is not able to distinguish meat from sugar by its visual impressions only, and remains in that state without any ulterior improvement.

These and many other new results which will be consigned and discussed in my forthcoming monograph, demonstrate that the cortical visual centre is not the seat of sensations of perceptions only; its function is to elaborate psychically the visual sensations which take place in the mesencephali ganglia, and more especially in the corpora quadrigemina, which must rank high in this respect according to well-established scientific facts.

Experiments on the monkeys confirm and throw fresh light upon this theory. The definitive visual symptoms presented by my monkeys after extirpation of the occipital lobes only, or of these and the angular gyri together, characterise clearly the psychical blindness in its opposition to absolute blindness and to simple amblyopia, and leave nothing to be desired from this point of view. When some time has elapsed after the extirpation, their visual sensations become perfect again; they are able to see minute objects, what they want is the discernment of things and a right judgment concerning their properties and their nature; they are deficient, in a word, of visual perception. For instance, if small pieces of fig, mixed with pieces of sugar, are offered to them, they are incapable of choosing by sight only, but require to take the sugar in their hand and put it in their mouth in order to assure themselves that it is not an alimentary substance. These are phenomena of psychical incomplete blindness, because the mutilation of the centres of visual perception has been incomplete. If the theory of Munk has been correct, that not only perceptions but also sensations take place in the visual sphere, our monkeys should have shown a deficiency not only in the former but also in the latter.

III. What we have said concerning the centre of vision greatly facilitates the comprehension of all which relates to the other sensorial centres; for one may expect *à priori*, and with much probability, that the latter are constructed on the same general plan as we have just seen obtain for the former.

With reference to the *auditory centre*, our experiments teach that no evident disorder of hearing follows extirpations of the frontal and occipital lobes in the dog. If, however, we remove the zone intervening between the temporal and occipital lobes, undoubted evidence of auditory disturbance is obtained, leading one to suspect that the limit of the cortical auditory centre merges superiorly into the portion of the visual sphere, which corresponds to the external segment of Munk. The effects of extirpations in the province of the parietal lobe confirm what was recognised by Ferrier, and afterwards by Tamburini and myself, that the bend of the third external convolution certainly makes part of the auditory sphere in the dog, but demonstrate also that this sphere irradiates into the parietal zone above this convolution, into the region corresponding to the zone F of Munk.[1] The fronto-parietal tract also, which extends in front, above and below the fissure of Sylvius, and comprises portions of the 4th, 3rd, and 2nd external convolutions, and the lower and anterior portion of the gyrus hippocampi, certainly contain an important portion of the auditory centre; and their destruction, we find, produces marked disturbances of hearing. Our results tend to show that not only the whole cortical area of the temporal lobe, as admitted by Munk, but probably also the cornu ammonis (which anatomists consider as an introflexion of the cortex) forms an integral portion of the auditory sphere. Thus we may conclude that the auditory area irradiates from its central point in the temporal lobe, upwards towards the parietal region, forward towards the frontal, backwards toward the hippocampal, inward towards the cornu ammonis.

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[1] See illustration representing Munk's Localisations, 'BRAIN,' Vol. II. p. 236; and Ross, 'Diseases of the Nervous System,' 2nd ed. vol. ii. p. 356.

With reference to the question as to the relation of the two cortical centrés to the peripheral organ of hearing, my new experiments confirm more fully what Tamburini and myself had already recognised; each ear having connections with both auditory spheres, but chiefly with that of the opposite side. In fact, every unilateral extirpation of sufficient extent in the province of the auditory sphere causes a bilateral disorder of hearing, more marked on the opposite side; and every extirpation in the same province made on the opposite side, after the effects of the first operation have ceased to be apparent, does not only set up disturbances in the other ear, but reproduces also those which had disappeared from the ear on the same side.

These phenomena are not explainable otherwise than by admitting that the same conditions obtain in the case of the acoustic, as do in that of the optic nerve. In both we must distinguish a crossed and a direct fasciculus; the former consisted of a much larger number of fibres than the latter. Neither of these fasciculi possess any uniform relation with distinct segments of their respective cortical spheres, but their fibres irradiate themselves throughout the area of those centres. The schematic figure B represents the extension assumed by the auditory centres on the external aspect of the hemispheres; and the relationship of the crossed and direct fasciculi of the acoustic nerve to the cortex is indicated by the dark and light dots respectively. A comparison between the figures A and B shows how, from the superposition of the visual and auditory spheres, there results an intimate connection and union between the two, embodied in an intermediary zone in which the "engrenage," or working into one another of the two centres, takes place.

There now remains the question of the functional nature of the auditory sphere; we must determine whether this centre be destined, like that of vision, to *perceptive* action only, or whether it be one of acoustic *sensation* also, as Munk affirms. The effects of more or less extensive unilateral extirpations within the limits of the auditory sphere last, according to our recent results, for a longer or shorter time according to amount of cortical matter removed, but always disappear after some time, or at least cannot be discovered by the means at our disposal. The symptoms consist in a more or less profound blunting of the acuity of hearing, but which never reaches complete deafness, and gradually fades off. An incomplete psychical deafness only subsists, by which the animal seems not to appreciate the meaning of sounds, noises, or calls, though it appears to hear them. The effects of a bilateral extirpation are always more serious: at first the auditory troubles may amount to absolute deafness, but soon pass into a condition of obtuseness of hearing; this also becomes attenuated and leaves behind it only more or less evident signs (according to the extent of the injuries) of psychical deafness. It seems therefore that Munk, in admitting a cortical deafness as a consequence of the bilateral extirpation of his spheres, is fallen into the same error which we alluded to with reference to the visual sphere, of attributing not only perceptive but also sensitive functions to the cortical centres. .

IV. My new experiments on the *olfactory* centre or sphere are much less complete; but still, by collating those in which disturbances of the sense of smell were observed, we may gather some notion concerning the position and extent of the corresponding cortical area.

Extirpation of the occipital lobe never gives rise to disorders of olfaction; destruction of the temporal lobe, or rather of the zone F of Munk, on the contrary, is followed by such. This fact shows that this region contains considerable prolongations of the olfactory centre. No evident deficiency of smell follows the extirpation of the temporal lobe; but if the lesion extends to the neighbouring convolution above the fissure of Sylvius one observes a notable diminution of that sense. Finally, a number of experiments show that a more or less extensive decortication of the gyrus hippocampi, as well as a partial ablation of the cornu ammonis produce olfactory disorders, at first an almost complete loss of smell—a fact which seems to indicate to us that this portion of the brain is the central point of the olfactory sphere. Some of my experiments show, in addition, that the more or less extensive destruction of the gyrus hippocampi, as well as the partial ablation of the cornu ammonis, produces evident troubles, and chiefly an almost absolute loss of olfaction, a fact which seems to show us that we have here the true centre, or fundamental nucleus, of the olfactory sphere.

These experiments, besides proving the great importance of the cornu ammonis in this respect, seem to point to a partial inter-hemispheric decussation of the olfactory fibres, analogous to what we said of the acoustic nerve—with this difference, however, that an opposite condition would prevail here, the direct appearing to be larger than the crossed fasciculus. Fig. C gives a schematic representation of the olfactory sphere, according to these views; but it shows only the part of it which emerges from the base of the hemisphere (hippocampal region) towards its external aspect. The region extends from below the Sylvian fissure upwards and forwards, and spreads out as far as the parietal lobe of the hemisphere, and to a less degree towards the frontal lobe. The dark dots, which are more numerous, indicate the relations of the direct fibres with the cortex; the light dots, present in smaller number, those of the crossed fibres.

We cannot state much concerning the localisation of the question for the sense of taste. The insufficiency of the means of testing it in animals, and possibly the hidden position they occupy on the cortex, may explain this want of success. Among our numerous researches we only once succeeded in proving a disordered condition of taste. After a unilateral extirpation of the fourth external convolution, and of a portion of the gyrus hippocampi, we discovered a diminished sensibility to the taste of bitters (powder of digitaline) on the half of the tongue corresponding to the lesion, as compared with the opposite half. From this fact we can only draw the presumption, that the gustatory is to be found in close relationship with the olfactory centre. And in fact, in the same dog, we were able to note the deepest alteration of the sense of smell.

V. My studies concerning the localisation of the *tactile* sense, or in other words concerning the *general sensorial sphere,* though still unpublished, were made in greater part in conjunction with Dr. G. Seppili, now physician-in-chief of the Asylum at Imola. Our results may easily be resumed as follows: consequently to the total or partial extirpation of the motor zone in the dog and the monkey, one constantly observes effects not only of motor paralysis, but also of cutaneous and muscular anæsthesia. When the lesion is not very extensive, one may, within a few days, obtain an almost perfect compensation of the paralysis of motion and sense; but when it involves the whole motor zone, or even if it is deep enough to reach the opto-striate tract, the phenomena are persistent, and one may have clear evidence of them (though less marked than at first) several months after the operation. After decortication of any one region which responds to electrical excitation with reactions circumscribed to a single part of the body, the sensori-motor paralytical effects are not limited to that part, but are diffused more or less to other parts of the body on the same side. Thus after extirpation of the motor zone of the posterior limb, there is a diffusion of the paralytical effects into the anterior limb, and *vice versâ;* after extirpation of the excitable zone of the anterior limb and face, or of the face only, or of the neck and trunk, there is diffusion throughout the same half of the body. We have already asserted, that these effects are more intense in that portion of the body of which the cortical area has been chiefly or solely removed. Thus, to quote but one instance, after decortication of the excitable area of the anterior limb only, the paralytical phenomena, though they extend into the posterior limb, are mainly localised in the anterior.

From these results we must necessarily conclude, that a localisation or precise delimitation of single centres in the sensory-motor zone, based upon not their reactions to electrical excitation of various points of the area, as Hitzig and Ferrier were the first to do, but upon the effects of partial extirpations, as Munk pretends,—that such a localisation is not practicable. We are led to believe that this is due to the fact, that the single centres in the sensory-motor zone are so completely bound up with, and, so to speak, let into, one another, that it is not possible to divide them with a clear and definite line, such as is the case when the cortex is incised and removed; so that in destroying a centre one necessarily eliminates a portion of the neighbouring centres.

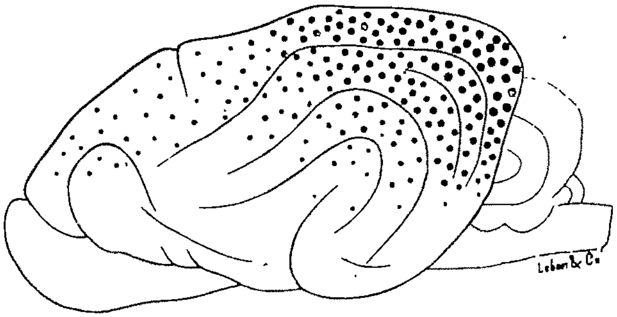
The almost perfect compensation of paralytical effects consecutive to the extirpation of a single excitable area, and which takes place after a few days, even in the part of the body corresponding to it, is a fact which argues in favour of the partial overlapping of the special centres contained in the general sensorial sphere. We must not forget the fact, demonstrated by myself in previous papers, that the centres of the limbs in the dog are not limited to the external cortex of the sigmoid gyrus, but extend over the whole still wider surface of the sulcus cruciatus, and it renders very difficult the complete extirpation of a single one of these centres. Considering the general sensory sphere as a whole, there remains the question whether it is limited to the frontal lobe, or whether it spreads out more or less into the other cerebral lobes. My recent researches exclude the occipital and temporo-sphenoidal lobes, but show that it does extend into the parietal lobe, extirpations of which constantly give rise to more or less extensive and durable tactile paralyses or pareses.

Let us remark finally, that in all experiments upon the tactile sphere there was a manifest and constant crossing of the relations between the peripheral sensory fibres and their respective cortical centres. Fig. D represents schematically the probable extension of the general sensorial sphere on the external aspect of the brain of the dog, comprising the several sensory-motor centres for the various parts of the body. One sees that what one calls "motor zone" is the central focus of the large portion of the sensorial sphere visible on the external aspect of the hemisphere—focus from which the latter is irradiated forwards towards the tip of the frontal lobe, backwards towards the parietal lobe, entering thus into intimate connection by "engrenage" with the visual, auditory, and olfactory spheres.

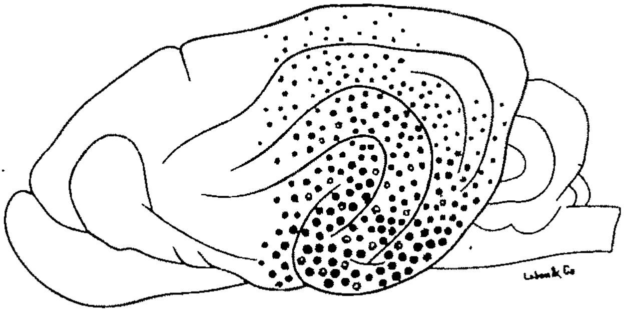
VI. If we compare together the four figures which indicate the localisation of these centres in the cerebral cortex, and ideally superpose them upon one another, we discover a remarkable fact, which I consider as one of the newest and most important results of my new inquiry. The four sensorial spheres, besides possessing each a territory of their own in the cortical substance, have, in addition, a common territory which is represented by the area of the parietal lobe, or more precisely by the zone F of Munk, which the latter calls the region of the eyes, attributing to it the humble functions of providing for the tactile sensibility of the eyeballs and conjunctiva.

In this region the mutual overlapping, or "engrenage," and consequently the partial fusion takes place of the single sensorial centres (excepting the gustatory centre, of which we know but little). Every one will thus see how this region is the most important in the hemisphere of the dog, where it represents, so to speak, the centre of centres. And indeed extirpation of this region, whilst it affects more particularly the visual perceptions, injures at the same time the auditory, olfactory, and tactile perceptions. There is no other portion of the brain of the dog lesions of which are capable of giving rise to effects so complicated, and so liable to set up profound psychical disturbances in the animal.

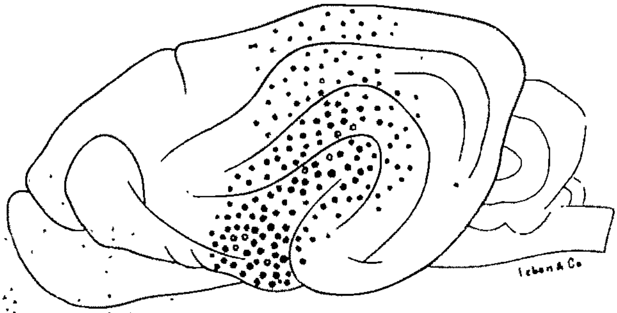
And in fact, after extirpation of the parietal lobes, Goltz—this diligent and able observer, but who starts from a point of view totally opposed to ours—has noted a notable alteration in the character of the animal; good-tempered and affectionate before the operation, they become peevish, quarrelsome, and refractory. He gives no explanation for this fact (which I have confirmed in a dog), and could not possibly give it on the grounds of his well-known theory. To me, on the other hand, this deep alteration in the psychical characteristics, appears to be referable to the loss of this important cortical area, in which the several sensory spheres meet and coalesce, and the removal of which necessarily produces the loss of the normal association of the psychical perceptions and images.



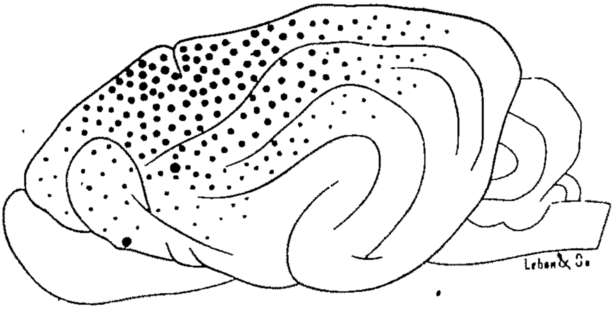
A.—VISUAL SPHERE.



B.—AUDITORY SPHERE.



C.—OLFACTORY SPHERE.



D.—TACTILE SPHERE.

To face p. 160.

THE RELATIONS OF HYSTERIA WITH THE SCROFULOUS AND THE TUBERCULAR DIATHESIS.

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(*Concluded from* Vol. VII., *p.* 28.)

CASE XL. (condensed). (Observations of Raymond, 'Gaz. Médic.' 1881, p. 431.)—*Tubercular Heredity. Pulmonary tuberculisation. Hysteria beginning with exaggerated and obstinate sweatings.*

Heredity.—Sister died of phthisis.

History of patient.—Locksmith (25 years) is admitted for a bronchitis of three months' duration. Dulness at the apex of the right lung (infraspinous fossa). Expiration prolonged and jerky, and dry crepitation. Very abundant sweating during the night (not on the hands). Usual treatment of tuberculosis, and granules of atropia. After four days the generalised sweatings have disappeared, and their place has been taken by sudoral secretion of the palmar aspect of both hands; the secretion is very abundant, constant, and increased by the slightest emotion. Failure of atropia internally; transitory success when in local hypodermical injections; success more rapid and more permanent with Duboisia. However, the sweatings reappear, and resist a series of other treatments. The mind becomes impressionable, irritable, anxious. He is moved by nothings, cries easily, and presents various nervous phenomena. Epigastric constriction, strangulation, repeated hysterical attacks. Right hemiplegia and hemianæsthesia, with contractions, aphonia, etc. "The general health in the interval of the attacks is perfect."

This case has the great advantage of being recent, and of having been followed with the greatest care. It therefore establishes incontestably that in a patient hereditarily tuberculous, there have been, first of all, typical signs of the pulmonary tuberculosis; that those lesions, instead of progressing (as it is the custom) have stopped, and probably even retrograded; and that, in their stead, a hysteria quite typical and complete has developed itself.

Let us notice, before passing on, the fits of sweating which appear here as in our patient in Case II., and which are useful to show, so to speak, the transition between the period of the pulmonary and that of the neurotic symptoms.

Case XLI. (condensed). (Brousse, 'Gaz. Hebd. of Montpellier,' 1881, p. 421.)—*Hereditary neuropathic antecedents. Personal scrofulous antecedents. Pulmonary Tuberculosis. Convulsive Hysteria.*

R., a soldier, admitted June 29, 1880, into the hospital St. Eloi.

Brother died of convulsions; sister has curious nervous disturbances at each menstrual period.

He looks delicate, rather effeminate; has shown in his youth evident signs of scrofula (scabs on the head, repeated blepharitis, etc.); when four years old violent measles, which leave after them a particular susceptibility of the respiratory apparatus. Since that time the patient is easily affected by bronchitis; he takes cold almost every winter, and at such times expectorates freely; expectorations often sanguineous.

Since ten years of age, convulsive attacks hysteriform in character: "First feeling of weakness in the lower limbs (he then knows that his attack is coming, and can save himself from a dangerous fall), then sensation of abnormal weight in the epigastric region, which from there goes upwards to the neck and seizes him at the throat; then beginning of the attack properly so called by a fall (without the cry), with loss of consciousness, afterwards clonic and sometimes tonic convulsive movements. After a few minutes the attack ends, the patient opens scared eyes, sometimes begins to cry. Soon after he is seized with a very acute feeling of fatigue, shuts his eyes and sleeps for many hours."

At his admission into the hospital, the sensibility of the whole body is noticed to be obtuse, with some hyperæsthesia, painful vertebral spots, reflex hyperexcitability, etc. In the chest signs of tuberculosis beginning in the apex of the right lung; a few dry crepitations, with harsh respiration and prolonged expiration in the right supraspinous fossa. In the heart there was a certain amount of stenosis of the pulmonary orifice.

Attack, during the visit of June the 30th. "Whilst he is being auscultated, R. suddenly gets pale, loses consciousness, exerts himself as if he wanted to lift up a weight which, starting from the epigastrium, seems to rise up to the neck and to suffocate him; afterwards appear clonic movements in the limbs and especially in the fingers; at the same time movements of translation of the body to the right and to the left, convulsions of the eyes upwards and contractions of the pupils. After a few minutes the patient regains consciousness, but the look is scared; the pupils are very dilated; the patient feels exhausted, and soon falls into a deep state of sleepiness, without stertor. During attack there have been neither froth at the mouth, nor biting of the tongue, nor even turgescence of the face." Similar attacks occur repeatedly. Dysentery.

On the 15th of August, the pulmonary lesions noticed when he was admitted have made no noticeable progress.

On the 1st of September the patient is dismissed, and leaves the hospital in a better state of health than when he entered it, without however being completely cured.

In this case, we have no alternations between the thoracic and the neurotic symptoms. The two orders of diathesic manifestations exist together. Only the very slow progress of the tuberculosis is to be noticed, as well as the slightness of the pulmonary lesions, although the patient has been examined when 21, and that since 4 years of age (measles) he has had frequent bronchitis and spitting of blood.

CASE XLII. (personal). (Condensed from the notes of MM. Brousse and Sarda.)—*Hysterical phenomena. Progressive Pulmonary Tuberculosis. Death and Autopsy.*

Virginie M., 47 years of age, admitted at the General Hospital the 28th of July, 1879.

Mother of a nervous temperament, died dropsical when 74. Rheumatic father. Nothing to note about brothers and sisters.

Courses appear when 17 years old. After six months disappear after a bath. At that time violent frontal cephalalgia, terebrant (like clavus); pains at the apex of the head; insomnia; fanciful appetite. Character sad, melancholic, sometimes excessively merry; frequent gastric cramps. Soon after, sensation of globus hystericus starting from the abdomen. The courses are re-established after five months. Paludean fevers repeatedly.

Married at 25, has nine labours or abortions. Frequent amenorrhœa, often intractable, brought on by emotions. After the death of one of her children (three days after birth), the nervous state grows worse. More intense and more frequent cephalalgia as well as sensation of globus; menstrual disturbances. At that time, persistent spot (for 10 months) of painful anæsthesia on right side of the thorax. Treated in succession in various wards of the hospital St. Eloi, sometimes for a metritis, sometimes for hysterical phenomena (peritonism, insomnia, various neuralgiæ, profound depression, etc.).

When admitted into the hospital, persistent pains more or less vague and erratic in character; state of hystericism without well-defined symptoms. All these neuropathic phenomena resist every kind of antispasmodic treatment. At the same time, dry, nervous, intractable cough. In November 1879, dulness in the right apex, in front and in the back; in the same region, prolonged expiration and respiration, harsh, even tubular, specially in front. The pains continue, however, to be very bad.

On the 15th of December, moist crepitations under the right clavicle. Local temperatures, 37· 2 (C.) under the right clavicle; 36· 7 (C.) under the left clavicle; 37· 2 (C.) in both axillæ. Always insomnia and considerable neuropathic malaise.

On the 14th of January, 1880, the pulmonary lesion has made alarming progress. Sibilant rales in the left apex; tubular breathing, humid crepitations, and cavernulous rales under the right clavicle. Greenish and very abundant expectoration. General state, always bad; much pain. Thrush in the mouth.

February 1st.—Signs of cavities under the right clavicle. Prolonged expiration, and harsh inspiration (without rales) in the left apex.

5th.—Delirium; pains, and moaning.

8th.—General state grows from bad to worse. The subcrepitant rales invade both lungs, the pulse very frequent becomes filiform. Progressive dyspnœa. Bronchial and trachæal rales, profuse sweatings, etc. Died on the 10th.

Post-mortem.—Right pleura thick and adherent on the whole extent of the middle and upper lobes, cavity in the apex communicating with a series of smaller cavernules. Grey granulations in the lower lobe. Analogous lesions (very advanced too) in the left lung. Passing over the other details of the autopsy (made however very carefully), let us merely mention four cysts in the broad ligament.

In this patient the hysterical phenomena have not been so marked as in the other observations. However, there has certainly been a state of obstinate neuropathy, a true hystericism.

The pulmonary tuberculosis has begun late, after a long period of purely nervous phenomena. It has rapidly taken the first place, and without entirely displacing the painful neuropathic state, has killed the patient in a short time.

This is the opposite of the cases in which the hysteria was on the contrary very pronounced, whilst the phthisis followed a course of remarkable slowness.

CASE XLIII. (personal). (Condensed from the notes of M. Brousse.)—*Hystero-Epilepsy. Pulmonary Tuberculosis.*

Marie F., 55 years old, admitted into the General Hospital the 11th of March, 1880.

Father died of old age when 94, is said to have had in his lifetime dartrous eruptions. Mother died of a pneumonia when 58. One of her brothers suffered from incontinence of urine during his young age.

She has had small-pox when 6 years old, and an erysipelas of the leg when 9. Often dartrous eruptions. The courses, which have begun when she was 22, have always been scarce and of short duration. Married; two children in good health. When about 40, had jaundice and lost her hair. Menopause at 48. (Frequent headache before the courses showed themselves; has been bled many a time.) Nervous temperament. Frequent headaches, vertigo before the attacks.

Beginning of the present disease (attacks) about 7 years ago. One evening after having quarrelled with her husband, she goes to bed, and during the night she has an attack, during which she passes her water, and strikes her husband. She has had many hæmoptyses, the last about 7 or 8 months ago.

During a previous sojourn at the hospital St. Eloi she had undergone an anti-syphilitic treatment. Hystero-epileptic attacks have been observed there, some of which disappeared by compression of the ovary. In her attacks, at the beginning of which she shouts loudly, she loses consciousness entirely, falls (sometimes she hurts herself in falling), foams, passes her water, etc.

At the present time she complains of numbness in the right side, especially in the right hand; she often pricks herself without feeling it; cannot work long with that hand, which is weaker than the other (22 with the dynamometer on the right, 25 on the left; she was not left-handed). Notable diminution of the sensibility (general and special) on the right side: the sight is even almost completely lost on that side. Frontal cephalalgia usual. A blister brings back a certain amount of sensibility. (I need not insist here upon the details of this æsthesiogenic action, which I have studied elsewhere.)

The 22nd of March one finds in the chest, under the right clavicle, externally, very plain dry crepitations during inspiration, with a slight dulness at the same level. Expectorates the following day sanguineous mucous matters, even at times pure blood. (She has had similar expectorations for about 8 months, and sometimes without any cough.)

29th.—Right ovarian pain under pressure. Sub-dulness under the right clavicle. Slight humid crepitations in upper third of that side. From time to time, dry crepitations under the left side. Behind, and on the left, dry crepitations also from time to time. Behind and on the right, slight humid crepitations less marked than in front. Obscured respiration in the basis of that side. Expectoration fluid, yellowish, tinted with blood.

Besides the major attacks she sometimes suffers from vertigo. If she is going about, she is obliged to stop and to sit down; if she is doing something she is incapable of going on, not knowing any longer what she ought to do.

It is an open question whether it is hysteria or epilepsy that we have to deal with in this case. In putting together all the details of the case (the hemianæsthesia specially), I incline rather to look upon it as one of hystero-epilepsy, that is of epileptiform hysteria. This being granted, we have another example of the coincidence of thoracic and neurotic manifestations in the same subject. It is the neurosis which predominates; the pulmonary tuberculosis began later, remained in the background, and progressed very slowly.

Case XLIV. (Tartivel, 'Union Médic.' 1864, I. p. 98.)—*Tubercular Heredity. Hysteria (cough; anæsthesia). Pulmonary Phthisis. Gradual disappearance of the spasmodic character of the cough.*

L. M. C., florist, age 20.

Father died of phthisis when 22, "after an illness of 18 months which it is impossible to mistake from the information given by the patient: heat and cold, neglected catarrh, repeated hæmoptyses, loss of flesh, continual cough, abundant expectoration, marasmus, death." Mother, on the contrary, strong and robust.

Sudden beginning with a violent fit of coughing, which lasted many minutes, threatened to suffocate her, and greatly frightened her. Was quiet for a few days. Then, after having been upset, new attack, new intermittence, and so on. At the same time abundant sweating in the palms of the hands. Cutaneous sensibility very much diminished on the left side, in the upper and lower limbs, on their external surface. Perversion of the capillary circulation: mottling of the extremities and hypothermy; sensation of dead fingers. Nothing is found in the chest, and the diagnosis is made of hysterical cough.

After a few months only the tubercular lesions appear in the left apex, and then the nervous phenomena improve gradually. After a period of fugacious, intermittent attacks, the thoracic phenomena entirely replace the neurotic. At that time, "on the right, in the whole height of the chest, respiration sounds very much weakened, incomplete, and mixed with rare disseminated, subcrepitant rales, more numerous at the base. No marked vocal resonance; weakening of the plenimetric resonance in the whole surface, especially in the lower third. On the right, harsh supplementary respiration, almost tubular. Expectoration rare, evidently purulent. These various symptoms are such, the author concludes, that it is impossible not to recognise now the tubercular infiltration of the left lung, with symptomatic hyperæmia."

Tartivel admits that doubts may be thrown upon the hysterical nature of that cough. However, the various characters of which he reminds the reader, and which he brings into connection with the sensory disturbances, seem clearly to indicate that the functional or neuropathic element has played a predominating part throughout the first portion of the history of the patient, whilst in the second phase, the anatomical or pulmonary element has come to the front and gradually displaced the former.

III. Summary and Interpretation of the Facts.

The 44 cases adduced, naturally fell under two categories. The first contained those (25 cases) in which the neurosis was the only manifestation of the tubercular diathesis; the second those in which both the nervous and pulmonary symptoms occurred either at the same time, or in succession.

The first group shows the filiation in the same family of tuberculosis and hysteria; the second allows in addition to study the order of succession of the two sets of symptoms, and their mutual influence upon one another. This group may be thus subdivided. With reference to the order of succession, three cases have presented themselves; 1, hysteria was manifested first, pulmonary troubles later (Cases XXVI., XXIX., etc.); 2, the opposite order was observed (Cases XXX., XXXI., etc.); 3, the two orders of symptoms occurred simultaneously, or nearly so (Cases XXVII., XXVIII., etc.).

Leudet, in an important paper ('Comptes rendus de l'Ass. franc. pour l'Av. des Sci.,' 1877, p. 829), comes to a similar conclusion: "Hysteria may precede, accompany, or appear in the course of, tubercular phthisis." He has seen only two cases in which hysterical followed pulmonary symptoms; a greater number of such instances will be found in the present paper.

With reference to the mutual influence of the two orders of symptomatic manifestations, a certain number of our observations show clearly a distinct alternation, the pulmonary symptoms taking the place of nervous symptoms and vice versâ. This substitution of one group of phenomena for the other may take place once in the course of the disease; or may, as we have also seen, occur several times.

When both orders of phenomena have had a simultaneous origin, this alternation is not observed; their evolution is synchronous, but even then there is evidence of their reciprocal influence, the pulmonary phthisis, for instance, being remarkably slow.

The final mastery remains sometimes to the one, sometimes to the other; we have mentioned cases where the hysterical, others where the pulmonary manifestations, definitively and exclusively conquered.

These propositions are more comprehensive than the conclusions of Leudet, with which however they are not in contradiction. He says: "Convulsive hysteria most frequently ceases on the appearance or in the first period of tuberculosis; . . . . its pre-existence does not prevent the rapid development of phthisis, but usually the latter runs a protracted course, and with longer periods of remission."

Let us now discuss the interpretation given to the facts by other authors, and bring this paper to a close with an endeavour to establish our views on a clinical basis.

1. It must be borne in mind that we do not deal here with two particular groups of facts upon which authors have much insisted, but form no part of our subject matter, viz., the pseudo-phthisis of hysteria, and the hysteriform symptoms of cerebral tubercle (pseudo-hysteria of phthisis).

With reference to pseudo-phthisis, it is well known that the neurosis may itself give rise to hæmoptysis, which if accompanied with loss of flesh and bronchitis, simulates the existence of pulmonary tuberculisation. Certain authors (e.g., Huchard and Mossé, loc. cit.) tend to refer to this category of cases all the instances adduced of phthisis and hysteria coexisting in the same subject. We admit that some cases may be objected to on this ground; thus for instance, certain observations by Largaud are not free from suspicion, and we mentioned them under all reserve. But let us not exaggerate, nor build up a whole theory on such slender grounds.

After eliminating all doubtful cases, a large number remain in which the phthisis was perfectly manifest, and in some confirmed by autopsy. Further, we adduce cases in which hysteria and consumption occur in the same family, but in different members. Hence it must be admitted that, making all allowance for, and putting aside pseudo-phthisis, there are certain relations between hysteria and true pulmonary phthisis.

We must likewise guard against the pseudo-hysteria of phthisis. Leudet and others have shown, with reason, the necessity of distinguishing the nervous phenomena arising from chronic diseases, and phthisis in particular; and we have endeavoured to keep this distinction before our eyes. We have, on the one hand, insisted upon the oscillations between the two orders of manifestations; on the other, we have carefully excluded cases where cerebral tubercles occurred.

2. The next explanation of the facts is the theory of the simple coincidence—a negation, not a solution, of the problem.

Thus Brachet will never see in pulmonary phthisis a true complication of hysteria, and with him many authors who adhere to the view formulated quite recently by Debove[1]:

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[1] 'Leçons cliniques et thérapeutiques sur la Tuberculose parasitaire,' Paris, 1884.

"Considering the great frequency of phthisis, there must frequently occur patients whose fathers or mothers, or some relation, have died tubercular, without there being in the fact more than a coincidence. In other words, phthisis is such a common complaint, that if one wished to demonstrate by statistics its relationship to any disease, one would find among the ascendants or descendants of a large number of patients proofs of one's thesis." We recognise the importance of this argument, against which the present paper is wholly directed.

If, as recent exaggerations and distortions of pathological facts have led many to think, tuberculosis is not hereditary, and be contagious only; if the family history loses its importance in presence of the influence of the surroundings and of the facilities the subject offers for the introduction of parasites—then the occurrence of one or more consumptives among the ascendants of a hysterical person is of no greater consequence than that of a cancerous individual among those of one suffering from trichinosis. If, on the other hand, genuine clinical experience is right in attributing a very fatal influence to heredity in tuberculosis, if the family history throws important light upon individual facts (as Bouchard showed with reference to diseases from a slackening of the nutritive processes), if clinical medicine can no more isolate the patient from his ascendants and descendants than from his actual surroundings, if diathesis be not an empty name but expresses an unfortunately too real element of disease—if all this is true, as we firmly believe, then the cases collected in this paper will exclude the possibility of a mere coincidence.

Quite recently I have had under my observation two families whose history might well have been recorded along with the above cases. In the first, two out of eight children died from phthisis; two have very frequent and violent sick headaches; one is a convulsive hysteric, another suffers from attacks of somnambulism. In the second, with marked tubercular heredity, three out of six children died consumptive, one is insane, another hysterical. Shall we see here mere coincidences, establish no relationship between these morbid manifestations, and attribute to fortuitous circumstances the fact that those who have escaped pulmonary disease are neurotic?

No; they all labour under the same diathesis, the manifestations of which are pulmonary or nervous. To speak of mere coincidences is to go against general pathology and clinical medicine.

I believe that every physician who will carefully look about him, not in hospital, but in private practice, will be brought like myself to recognise certain relationships between hysteria and phthisis unexplained by the above-mentioned theories.

3. A different interpretation has been given, by Bernutz and Jolly, of the influence of the tuberculosis of parents upon the hysteria of children. "A much more serious objection," says the former ('Dictionnaire' of Jaccoud, p. 196), "may be raised against the opinion of Chairon, that pulmonary phthisis among the ascendants may be a predisposing cause to hysteria among the children . . . . this neurosis is much more common among those who have early been left orphans . . . . it is by depriving them of their father or their mother that phthisis favours the development of the neurosis among the descendants." The circumstance alluded to in this passage could, however, be called in to explain the development of tubercles, as much as that of hysteria in children deprived early of their parents and exposed to ill-treatment. Among our cases we find eight where the information necessary to elucidate this point is not given. In five others (II., VII., XII., XIII., XIV.), the view advocated by Bernutz might find some support; but in ten others it is inadmissible. Thus in Cases I. and III. the father and mother are living, and the child belongs to a good social position and is well cared for. In Case IV. the social position is high, and the disease appears at the age of 20; likewise in Case V. it manifests itself after marriage. The patient in Case VI. has been well brought up by the father and grandmother; in Cases XV., XX., XXIV., the neurosis appears late; and both parents of the patients in Cases XVI. and XXIII. are living. We look therefore upon the theory of Bernutz as insufficiently supported by facts.

According to Jolly (Ziemssen's 'Cyclopedia,' vol. xii.), it is not the fact of being an orphan early in life, but the congenital debility, which produces hysteria among the children of phthisical parents. "Various enfeebled conditions of parents, from chronic disorder or otherwise, tend to develop a hysterical disposition among the children. Such an influence has more particularly been attributed to phthisis."

We shall meet this theory with a diametrically opposite affirmation by Briquet: "Organic diseases of the heart, pulmonary consumption . . . . are not usually followed by hysterical symptoms, whatever pain and weakness they may cause . . . . hence we must conclude that a great perturbation upon which all the vital forces are concentrated, does not allow the development of the nervous orgasm in which hysteria takes its origin."

As a matter of fact, in families where the diathesis prevails, it is not those most debilitated by the hereditary taint who become hysterical, for they become consumptives of the common type. Those on the contrary who, at least temporarily, resist the pulmonary affection develope the neurosis.

4. In opposition to the theories which seek to explain hysteria by the antecedent tuberculosis, we have those which tend to connect the final tuberculosis to an anterior hysteria. Thus Bernutz (loc. cit., p. 274) admits that hysteria may favour "among predisposed subjects the development of certain chronic diseases, more particularly of consumption, which since F. Hoffmann has been generally considered as a possible (and I may say pretty frequent) consequence of the neurosis."

Briquet likewise says (loc. cit., p. 537): "It is easy to understand how Hoffmann, Louyer Villermay, and Georget, seeing the nutrition of hysterical people to be disturbed either through the want of a sufficient alimentation or through worry of constant pain, ascribed to this fact the production of tubercles. All authors, indeed, allow that phthisis may arise as a sequel of hysteria, and there are good reasons for adopting their opinion."

Other writers, however, have opposed this view. Thus Monneret and Fleury ('Comp. de Méd.' v. 79) say: "Tubercle has been considered as complications depending on hysteria. Its development may be favoured by nervous troubles impeding respiration, but it will be conceded that it is going too far to speak of phthisis as a result of hysteria." Brachet ('De l'Hystérie,' p. 383) also says: "Cheyne is mistaken when he says that phthisis was the result of violent hysteria." Huchard (loc. cit., p. 1056), too, considers it an error to say that hysteria favours the development of tuberculosis.

Finally Leudet (loc. cit., p. 831) answers these theories by the following statistics: "I said that 95 hysterical women had been admitted several times into my wards at intervals varying from 2 to 22 years. Now out of these 95, only 9 presented symptoms of pulmonary consumption. Out of 324 hysterical patients, only 23 showed signs of consumption. Hence I may conclude that phthisis is not common among the hysterical, and that the neurosis does not constitute a predisposition to organic lung disease."

We personally shall answer to those who will see in phthisis a consequence of hysterical enfeeblement, that this neurosis is, on the contrary, remarkable for the preservation of strength and resistance, which is observed even in cases where alimentation and nutrition are in abeyance. Further this theory does not explain the facts in which hysterical symptoms appear at a later date than the thoracic lesions, and still less those wherein hysteria is the only representative in the individual of the hereditary tubercular diathesis.

5. Many authors have been chiefly struck by the compensatory oscillations of the neurotic and tubercular manifestations, and have interpreted them as an antagonism of two diseases lying alongside, and have framed their views accordingly. Briquet states that he has seen phthisis "suspend the progress, and remove every symptom of hysteria" (p. 500). Tartivel found the same in the case we have quoted, and repeats that "*febris spasmos solvit.*" Féré ('Arch. de Neurologie, 1882,' p. 170) reminds us of the cases of the 'Iconographie' where hysterical fits and anæsthesia disappeared during the last step of pulmonary phthisis, adding that "it is impossible to decide therefrom whether it is the general exhaustion which brings about the disposition of the nervous symptoms, if whether fever is enough for that purpose, or whether there is some other cause at work." He eliminates the first alternative by quoting a case where a simple pleurisy suspended the hysterical condition, and the second by quoting one where acute articular rheumatism did not have that effect.

Pidoux ('Etudes sur la Phthisie') has noted the moderating influence of hysteria upon tuberculosis. "Neuroses," says he (p. 151), "hysteria, melancholia, the proteiform neuropathic state known as "nevrosism," often act as moderating agents. Phthisis in neurotic subjects has a very slow development, and periods of long intermission. I have no doubt that such subjects, among whom the disease is not rare, offer to it an extraordinary and almost indefinite resistance; and in treating them, we must not pay too much attention to their abnormal nerves." And again: "If a pulmonary consumption is combined with a neurosis, the organic lesion will evolve very gradually; gastralgias and enteralgias, have often this effect. I look upon this statement as an axiom."

Leudet (p. 831) quotes the opinion of Briquet and that of Walshe, which he nearly adopts: "Walshe ('Diseases of the Lungs,' p. 476) says, with reason, that phthisis and hysteria appear as a rule to be antagonistic; in other words, the pre-existing hysteria disappears when the pulmonary disease takes a certain development. . . . The opinion of these two authors is true for most cases; still there are exceptions."

Finally, Largaud (Thesis, Montpellier, 1882) has, under the influence of his master, Dr. Cavaillon of Carpentras, clearly brought into relief the antagonism of hysteria and tuberculosis considered as two distinct affections. "We shall show how consumptives, even when most threatened, may live, thanks to hysteria . . . . the neurosis appears to play the part of a protector, as if in its presence the pulmonary complaint had its symptoms mitigated and its progress arrested." And again: "Hysteria in a consumptive is not a symptom of tuberculosis; it is not a complication; it is only a new morbid state, without causal relation with it. . . . The only thing which may be affirmed is that hysteria acts as a safety-valve. . . . This is another illustration in support of the ancient aphorism of the Father of Medicine, *Duobus Doloribus*, etc." And finally: "The chief conclusion of our thesis is that consumptives may, if not recover, at least live for a long time, thanks to their hysteria. . . We shall therefore respect the neurosis as affording an element of safety to those patients. . . ."

We grant most readily to these authors the fact, observed by ourselves, of alternatives between pulmonary and neurotic manifestations; but we cannot conclude therefrom to a theory presenting the two states as distinct diseases, fortuitously co-existing in the same individual, and exercising a true antagonism upon one another.

First, if an alternation is common it is not constant, and we have given cases where no trace of antagonism could be discovered. Leudet also quotes cases showing that in the midst of hysteria, phthisis may make rapid progress instead of being arrested. All the facts observed by the authors mentioned in the two preceding paragraphs, and brought forward in support of the theory of a mutual pathogenic influence between hysteria and phthisis, are also opposed to the doctrine of their antagonism. The views against which we are writing on account of their narrowness and onesidedness, rest upon groups of well-observed facts, which are destructive of the conflicting generalisations drawn from them.

But in the second place, in order to admit the theory of their antagonism, we must assume that hysteria and tuberculosis are two complete and distinct diseases. Now, hysteria is a neurosis, and a neurosis is always symptomatic; it is a "syndromon," or collection of symptoms, and not a disease or morbid state. The theory, after all, rests upon the assumption of a fortuitous coincidence of hysterical with tubercular accidents; it excludes every hereditary filiation between the successive morbid manifestations, and is therefore liable to the objections raised in our second paragraph.

6. We may finally mention a last theory upheld by Huchard (loc. cit., p. 1073): "Among diathesic states pulmonary tuberculosis has been mentioned among the antecedents of hysteria. But it is only the rheumatic and arthritic diathesis which should be adduced as capable of giving rise to both tuberculosis and hysteria; the latter is in no way under the influence of the tubercular affection." We grant that arthritism may give rise to hysteria, perhaps even oftener than tuberculosis, but it is not a reason to attribute to it all the instances of the neurosis observed in tubercular families. If we analyse our cases from this point of view, we see that in the great majority it is impossible to find a proof to that effect.

We have reached the end of our critical review of the theories proposed, and are satisfied with none. Neither the pseudo-phthisis of the hysterical, nor the pseudo-hysteria of the tuberculous, nor a simple coincidence, can account for all the facts. We cannot say that the children of phthisical parents become hysterical on account of their inherited debility, or of their becoming orphans early in life; nor that hysteria predisposes to phthisis by debilitating the subject. We cannot admit of two antagonistic diseases, nor appeal to arthritism as a common source of the two orders of manifestations.

Upon the ground thus cleared remain standing the views held by the Montpellier School of Medicine, which we shall here briefly restate.

Tuberculosis, like all other diatheses, is an essentially general and constitutional disorder; pulmonary phthisis is but one of its manifestations. It may show itself as a neurosis, and more particularly as hysteria.

Just as chorea is often a rheumatic disease, angina pectoris a gouty disease, thus hysteria may be a tubercular disease, taking the word tubercular in its nosological, not in its anatomical sense. In speaking of hysteria as a tubercular disease, therefore, we do not assume the existence of tubercular matter in the nerve-centres, for we do not look upon tubercles as pathognomonic of the diathesis, which exists without them as they may exist without it.

The tubercular diathesis is essentially hereditary, but may be understood only if it be followed up in families under its various manifestations from generation to generation. We then see how hysteria may represent the diathesis in one of the members of the family as meningitis may do in another, or Pott's disease in a third.

Besides such cases (I.–XXV.), in which hysteria is the sole manifestation of the diathesis, there are others (XXV.–XLIV.) where the same individual presents simultaneously or successively the two orders of manifestations, pulmonary and neurotic. We then witness the typical phenomena which characterise other protean diseases: the two orders of symptoms alternate and replace one another; or if they be simultaneous, influence and modify one another, until one of them gets the upper hand. In other words, the mutual relations between the phthisis and hysteria of the tubercular diathesis are like those between the cerebral and abdominal symptoms of typhoid fever, or between the visceral and articular manifestations of gout.

These remarks concerning hysteria and phthisis form but a fragment of a great and true chapter concerning the relationship of all neuroses and even of all nervous diseases to the diatheses and general diseases.

If we cannot understand the neuroses without perceiving their mutual relations, if the consideration of the "*neuropathic family*" is indispensable for securing a clear grasp of the phenomena of neuropathology, it is yet necessary to go still further and complete all this by the conception of the "*diathesic family*," outside of which we shall find nothing but isolated, scattered, meaningless units.

One word in conclusion on the therapeutic applications of our theory. Hitherto, the neurotic element of hysteria has much too exclusively attracted attention; the diathesic element has not been met by treatment. Though we have no specific against diatheses (except in the case of syphilis), we nevertheless have various rational means to deal with them. The treatment of rheumatism is not that of scrofula, nor that of tuberculosis. Hence, it is not indifferent to know whether a neurosis depends upon the one or the other constitutional state.

Three categories of indications will have to be fulfilled in the full treatment of hysteria. First, the actual symptoms are to be met with appropriate measures, such as ovarian compression, ether inhalations, æsthesiogenous applications, etc. Second, the state of the nervous system, which is to be modified at any cost: hydrotherapy is often useful; change of surroundings; electricity, galvanic, but more especially static. We have little faith in the bromides. Third, the diathesis forms the fundamental ground for a chronic exhibition of arsenic, sulphur, alkalies, chloride of gold, mineral waters, etc.

Unless these three orders of indications be duly taken into account, our measures, based upon a superficial diagnosis, must necessarily remain sterile.

ON THE CONDITION OF THE FUNDUS OCULI IN INSANE INDIVIDUALS.

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(*Continued from* Vol. VII. *p.* 50.)

General Paralysis of the Insane.

We have examined 66 cases coming under this designation; most of them have been examined more than once, and many several times; in all, 135 separate examinations have been made. Of these 66 cases, 49 were males and 17 females; the average age of the patients was 40·28 years.

Of the 66 cases, 43 (65·15 per cent.) we considered to be normal; 15 (22·72 per cent.) were decidedly abnormal, whilst in 8 others the changes were so slight that we did not like to pronounce them positively abnormal; if however these be added to the 15 distinctly abnormal ones, we get a percentage of 34·84 per cent., under this latter category.

A qualification is here requisite; in one of the cases (No. 37 in table), the changes noted were (mainly, at any rate) due to Glaucoma, and are therefore foreign to the issue before us; a similar remark applies to two others (Nos. 36 and 62) in which the abnormalities present were due in all probability to the associated retinitis pigmentosa; it would, we think, be safer to exclude these cases, which would therefore leave 12 distinctly abnormal, to which if we add the 8 doubtful ones we should get a percentage of 30·30.

PLATE I.

Fig. 1

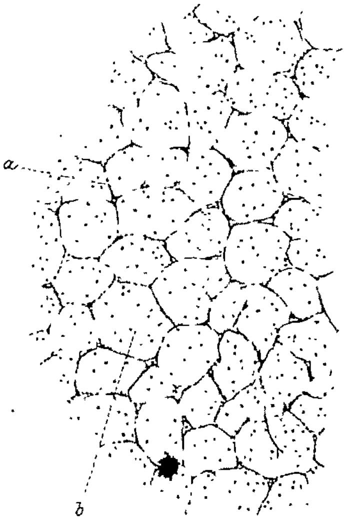


Fig 3

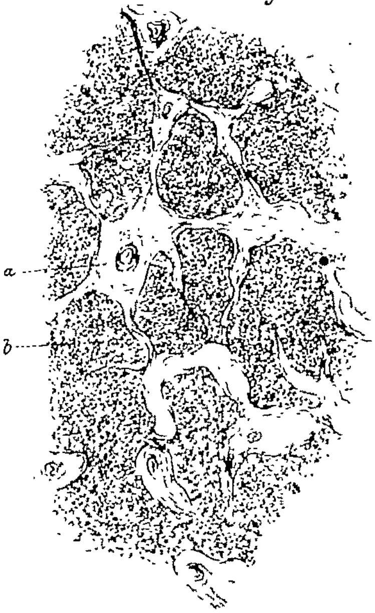
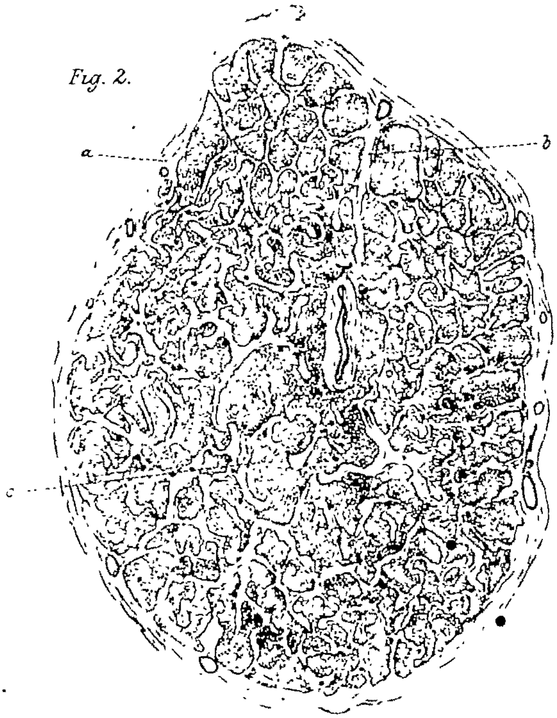


Fig. 2.



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DESCRIPTION OF PLATES.

PLATE I.

Fig. 1.—Transverse section of optic nerve, a short distance behind eyeball; from a case of General Paralysis of 3½ years' standing. (Case No. 23 in Table I.) Normal. × 50.

(*a*.) Normal trabeculæ.
(*b*.) Healthy nerve tubules.

Fig. 2.—Transverse section of optic nerve a short distance behind eyeball; from a case of General Paralysis. (Case No. 18 in Table I.) Atrophy; consecutive to slight chronic interstitial neuritis. × 50.

(*a*.) Thickened internal sheath.
(*b*.) Thickened trabeculæ.
(*c*.) Nerve tubules undergoing granular degeneration.

Fig. 3.—A portion of Fig. 2, × 300.

(*a*.) Greatly thickened trabeculæ.
(*b*.) Nerve tubules undergoing granular degeneration.

PLATE II.

Fig. 4.—Transverse section of optic nerve a short distance behind eyeball; from a case of General Paralysis. (Case No. 28 in Table I.) Atrophy; primary at disc. × 50.

(*a*.) Greatly thickened internal sheath.
(*b*.) Thickened trabeculæ.

Fig. 5.—A portion of Fig. 4, × 300.

(*a*.) Thickened trabeculæ.
(*b*.) Neuroglia corpuscles, and small round cells.
(*c*.) Nerve tubules undergoing atrophy.

(The artist has here depicted the best part of the section; in most places the trabeculæ are thicker and closer together than are here represented.)

Fig. 6.—Transverse section of optic nerve, close behind eye-ball; from a case of General Paralysis. (Case No. 42 in Table I.) Early cirrhosis. × 300.

(*a*.) Thickened trabeculæ with well-marked nuclear elements.
(*b*.) Hypertrophied neuroglia corpuscles.
(*c*.) Healthy nerve tubules.

PLATE II

Fig. 4.

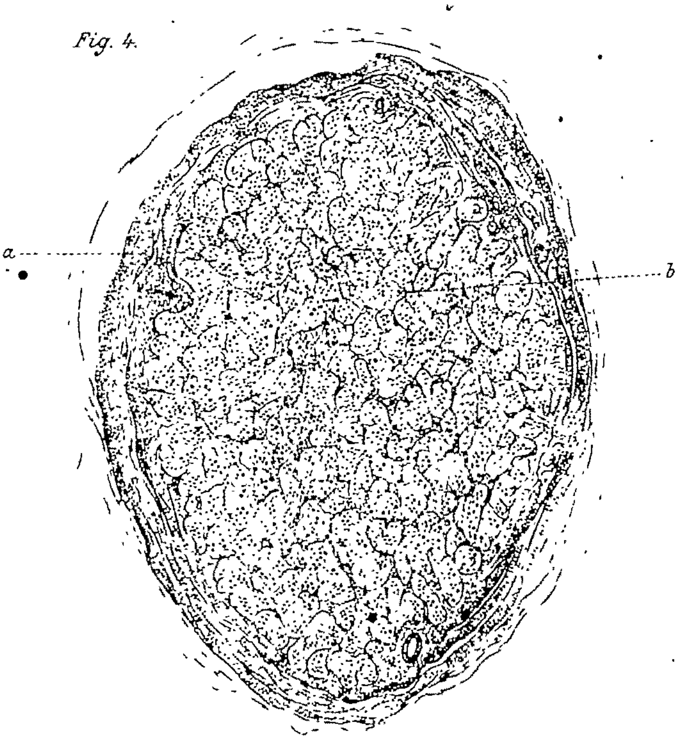


Fig. 5.

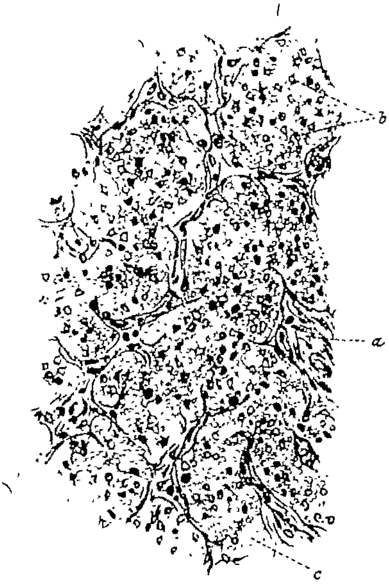
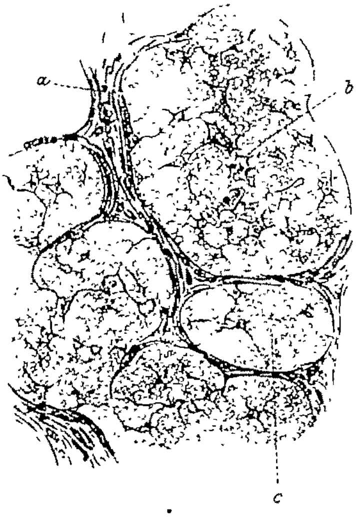


Fig 6.



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We append a table giving details of every case examined, and in the following account the numbers given refer to the numbers of the cases, in this table. (See pages 34 to 45.)

We have there divided the disease into stages, first, second, and third; the first being the early period of mental excitement and exaltation; the second dating from the subsidence of excitement, may perhaps be broadly characterised as one of advancing dementia, though at times patients improve very much at this period; whilst the third is the final stage of complete fatuity, during which the patient is quite helpless, and for the most part confined to bed. This is but a rough classification; the primary stage of exaltation is often absent; and there is no uniformity in any two cases, as regards the duration of the stages: nevertheless we have thought it well to include it, as giving at least a general idea of the period in each case at which the examinations were made. (It may perhaps be advisable to note that this division is not quite the same as that adopted by some authorities, who call the first stage, that of commencing mental alienation, prior to the advent of excitement, but the classification we have chosen we think preferable for the purpose.)

In 10 of the 66 cases the optic nerves and discs have been submitted to microscopical examination; the cases examined are indicated in the table, the details being given in the text.

It will be seen that four abnormal conditions of the optic discs have been met with. (1) Simple hyperæmia. (2) The same, combined with some amount of softening or blurring of the edges, the extent of this being never very great. (3) Simple anæmia. (4) Distinct atrophy. These are not however distinct conditions, but more or less different stages in one process, or we would rather say two, for we feel pretty confident that complete atrophy of the discs may occur without a previous stage of hyperæmia.

The cases in which decided hyperæmia of the discs occurred with more or less blurring of the edges are Nos. 3, 21, 34, 42, 13. In No. 3 the abnormal redness, &c., came on whilst the patient was under observation, and a microscopical examination of the parts involved was made after death, which took place two months after the last ophthalmoscopic examination.

In a transverse section of the optic nerve immediately behind the eyeball it was found that the trabeculæ, or fibrous septa between the nerve bundles, were thickened, in some parts decidedly so, in others scarcely at all: the neuroglia corpuscles were distinctly hypertrophied, sending out branching processes interlacing with each other; they also appeared more numerous than usual: these conditions were not uniform—some parts appearing healthy or nearly so. The nerve tubules seemed everywhere quite healthy. The conditions in fact were similar to, but less advanced than, those observed in case No. 42 (described below).

In No. 21 the discs when first examined were abnormally red, but this had subsided 2½ months afterwards, and these parts then appeared normal.

In No. 34 the discs remained in the hyperæmic condition during the six months patient was under observation, though the redness was subsiding when the last examination was made.

No. 42 was a good example of the process in question: the hyperæmia distinctly increased during the eight months the patient was under examination, and the edges of the discs were seen to become very misty and at length difficult to distinguish; the patient died two months after the last examination, of pneumonia, and a microscopical examination of the optic nerves and discs was made. It was found that the inner sheath of the nerve was scarcely at all thickened; but the trabeculæ were everywhere enlarged—in some parts very greatly so—the contained nuclei being very distinct; the neuroglia corpuscles were much hypertrophied, and their processes so numerous and distinct, that in many of the nerve bundles a distinct network of them could be traced with great facility; they appeared also to be increased in number. There was an absence of small cell infiltration in the nerve, but there appeared to be a little in the course of the fibres on the disc. The nerve tubules everywhere appeared quite healthy. (Plate II. Fig. 6.)

We wish particularly to refer to No. 13, which we think very interesting and important. This case was under observation 20½ months, and the process was watched through all its stages, from commencing hyperæmia, to distinct atrophy. On the first examination, there was a suspicion of abnormal redness of the discs; on the second (two months later) distinct evidence of slight neuritis was found; this remained stationary for about a month, and indeed underwent very little change for another six months, though the redness seemed to be very slowly subsiding; at the end of 13½ months from the first examination, the right disc had become distinctly paler though the left continued red, whilst in another three months there was a still further diminution of colour in each disc; in another four months (20½ months from first examination) the right disc had become distinctly atrophic, whilst the left still retained colour, though this was fast subsiding.

We thus had here an opportunity of watching through its whole course a process which more often comes under observation at isolated stages, and though the inference that the process described occurred might have been legitimately drawn from the observance of the discs, at these isolated stages, the case just quoted furnishes an exemplification of this, as complete as could be desired. We should be disposed to style the process a slight neuritis,[1] running a very chronic course. A microscopical examination of the optic nerves was made in this case. The internal sheath of the nerves was moderately, but not greatly thickened; the trabeculæ were generally thickened, in some places very greatly so, and presented for the most part a translucent, imperfectly fibrillated appearance; in the centre of the thickest parts, enlarged arteries could in most cases be distinguished; the neuroglia corpuscles were increased in number, and to a certain extent in size, but there was no distinct small cell infiltration; in a few parts, what with the thickening of the neuroglia and the great development of the trabeculæ, the nerve bundles appeared almost obliterated, and in some of the sections a distinct band of fibrous tissue ran from the periphery inwards, in which but slight evidence of nerve bundles could be found; the fibrous thickening was thus much more advanced in some parts than in others. The nerve bundles appeared everywhere to have undergone degeneration, their position being occupied by granular-looking masses, in which normal nerve tubules could not be distinguished. The nerves as a whole were much diminished in size. (Plate I., Figs. 2 and 3.)

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[1] This term is perhaps not free from objection, but the process appears to be a chronic interstitial inflammation,—a cirrhosis, in fact.

It seems worthy of note that in two (Nos. 3 and 13) of these five cases in which evidence of slight neuritis was found, well-marked pachymeningitis of the cerebral dura mater extending quite down to base was disclosed at the autopsy ; in two other cases however (Nos. 21 and 42) this condition did not exist ; whilst in the other case (No. 34), no opportunity was afforded of ascertaining this point.

But whilst atrophy of the discs may thus succeed a process which can only be described as a slight chronic interstitial neuritis, we have also evidence that it may independently occur ; but we must first notice a condition of anæmia of the discs of which Nos. 18 and 38 are examples. No. 18 was under observation for eight months ; the pallor of the discs came on under observation, and was so great as to suggest atrophy ; nevertheless this patient's acuteness of vision was perfect. We have before given it as our opinion, that if a patient can read J 1 and $\frac{20}{20}$ he has not as yet got atrophy, whatever he may be going to have. In the case before us, the pallor showed a tendency to diminish rather than to increase ; we were not able to trace its subsequent progress owing to the patient's removal from the asylum, and hence, although the case may not improbably have ended as one of atrophy, we have no proof that it did so. No. 38 was another case in point, though here we had no opportunity of comparing the appearances presented by the discs with a previously normal state.

We have marked as distinct cases of atrophy, Nos. 28, 44, 45, 53, and 59.

In No. 45 the appearance of the right disc was strongly suggestive of commencing atrophy. This patient had the clinical signs of sclerosis of lateral columns of cord.

The other four cases were well-marked examples, and two of the patients (Nos. 28 and 59) were quite blind. No. 28 was a very interesting case. The patient apparently completely recovered from the first stage of the disease (as to the diagnosis of which there was at this time a little doubt), and was discharged from the asylum; it happened that an ophthalmoscopic examination had been made, and nothing abnormal detected; nine and a-half months after the patient's discharge (about 14 months after the ophthalmoscopic examination) she was brought to the asylum by her husband, with the complaint that her eyesight was very bad, and had been failing since about a few weeks after her return home; both optic discs were then in an advanced condition of grey atrophy, and patient was nearly blind; her mental faculties were also clearly failing—she was in fact becoming demented. She was re-admitted into the asylum about 4 months subsequently, when the appearances presented by the optic discs were substantially the same, as they were also noted to be at the last examination, when patient was well advanced in the third stage of the disease.

A microscopical examination of the optic nerves and discs was made. It was found that the internal sheath of the nerve was very greatly thickened; the trabeculæ were also decidedly thickened and approximated to each other, forming in many places wavy bundles of fibrous tissue, the nuclear elements in these bundles being in many places very prominent. The neuroglia corpuscles were increased in number apparently, but not notably in size, whilst there was a general infiltration throughout the nerve bundles of small cells. The nerve bundles were much obscured by the thickened trabeculæ, and nuclear elements, and had clearly undergone considerable wasting, but could in most places be distinguished by careful examination. The nerve as a whole was much diminished in size. The transverse section above described, from this case, presented considerable difference as compared with that from No. 13; the chief points of contrast being that the internal sheath was more thickened, and the trabeculæ somewhat less so in the present case than in No. 13; the nerve bundles appeared also less degenerated in the former than in the latter, though possibly post-mortem changes may have been a factor in this; the nuclear elements throughout the section were also much more numerous in the case last described than in No. 13. (Plate II. Figs. 4 and 5.)

In No. 44 the atrophy, though not so extreme, was yet far advanced. This case had the clinical signs of locomotor ataxy; and after death grey degeneration of the posterior columns of the cord was found throughout the greater portion of their length.

No. 53 is still under observation; the first examination was made when patient was yet in the first stage of the disease, and well-marked atrophy was noted then, without any trace of hyperæmia; the atrophic condition appears to have been steadily advancing.

No. 59 is also still in the asylum; he is quite blind, and complete atrophy of his discs was found when he was first examined; the patient was however at that time well advanced in the second stage of the disease, and the appearances of the discs were quite consistent with the former presence of neuritis; in fact they rather suggested such an origin.

This patient has well-marked signs of sclerosis of lateral columns of cord.

As regards the question of a relation between spinal symptoms and optic nerve atrophy in General Paralysis, our cases furnish both positive and negative information. Of the five cases of optic nerve atrophy which we have described, one (No. 44) had posterior sclerosis, and two others (Nos. 45 and 59) signs of lateral sclerosis. In No. 28, on the other hand, than which no case could have been more typical, not only were spinal symptoms absent during life, but from a careful microscopical examination of the cord, we are able to declare that it was perfectly healthy, sections taken from the cervical, dorsal, and lumbar regions, presenting perfectly normal characters. In No. 53, the knee jerks are normal, and there is no ankle-clonus. On the other hand, No. 66 has signs of lateral sclerosis, while the discs are described as normal.

We think that in at least three of these cases, Nos. 28, 44, and 53, the atrophy was primary, that is, was not preceded by any prior stage of hyperæmia at the disc, but the patients not having been under close observation previously, it is doubtless impossible to affirm this absolutely. It may, however, be remembered that in No. 28 a previous examination had been made, though sufficient time had probably elapsed between this examination and the second, for a neuritis to have set in and run its course. In No. 53 again the discs were distinctly atrophic whilst the patient was yet in the first stage of the disease; in connection with which point it is not unimportant to observe, that those cases of slight neuritis which have come under our notice have run a very chronic course, remaining for months practically unchanged.

We ground our opinion, however, quite as much on the appearances presented by the discs at the time of examination, which were such as strongly to suggest that the changes were primary. The atrophy then would in all probability be due to destruction of nerve fibres by a chronic inflammatory process (similar to that we have noted at the discs in other cases) occurring higher up—in the intracranial course of the optic nerves—in the optic tracts—or even at the nuclei of origin; the nerve tubules below the seat of disease subsequently wasting.

In connection with this point it may be stated that in No. 28 the corpora geniculata had altogether disappeared, the corpora quadrigemina appearing normal to the naked eye.

The cases we have marked as doubtful are Nos. 1, 2, 22, 39, 43, 48, 51, and 60. We think it very possible that some at least of these cases were abnormal; in Nos. 39 and 60, for instance, the redness noted may not improbably have been the commencing hyperæmia of the neuritis previously described; whilst, on the other hand, in No. 43 the pallor may have been that of commencing atrophy; the changes being however so slight, we have preferred not to pronounce positively upon them. We may mention, however, that in No. 1 the blurring of the inner edges of the discs was probably a physiological condition, that was at least the impression conveyed by the appearance of the discs, which were perfectly normal in all other respects, and the condition in question has remained absolutely unchanged for 2¼ years.

It is unnecessary to comment upon the remaining cases which we have pronounced to be normal, except in the case of those, six in number, in which a microscopical examination of the optic nerves,[1] &c., was made after death.

No. 12. The last ophthalmoscopic examination was made

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[1] Sections in all cases taken close behind the eye-ball.

2½ months before death, the patient being at the termination of the second stage of the disease, which had lasted about six years.

The nerve tubules were healthy; the trabeculæ were not thickened, but the nuclei in them were unusually distinct, and there was a very slight increase in the number of neuroglia corpuscles.

No. 23. The last ophthalmoscopic examination was made three weeks before death, the patient being in the third stage of the disease, which had lasted upwards of 3½ years.

The optic nerves were perfectly healthy. (Plate I. Fig. 1.)

No. 24. Examination made two months before death, patient being in the third stage of the disease.

Optic nerves normal.

No. 27. Examination made a few hours before death, the patient being accidentally suffocated by food getting into his larynx, whilst yet in the second stage of the disease.

There was found a slight increase in the number of neuroglia corpuscles, with perhaps slight thickening of the septa here and there, but the nerves were otherwise normal.

No. 33. Examination one week before death.

Nerves for the most part quite normal, but in one or two places the trabeculæ appeared a trifle thick, and here also there was some slight increase in the number of neuroglia corpuscles.

No. 40. Examination made 3½ months before death.

Nerves for the most part perfectly normal, but septa perhaps a trifle thick in one or two places.

It will thus be seen that in some of the clinically normal cases there was found after death a tendency to increase on the part of the fibrous elements of the nerve, manifested by slight thickening of the septa, and slight increase in the number of the neuroglia corpuscles; the changes in question however were in all cases quite trivial.

By a perusal of the microscopical reports, particularly of course those of the abnormal cases, it will be seen that the pathological process is essentially one of increase in the fibrous elements of the nerve, at the expense of the nerve tubules; in cases Nos. 3 and 42, where the process was examined in its early stages, a luxuriant overgrowth of the neuroglia elements was found, with thickening of the fibrous tissue framework, the nerve tubules being as yet perfectly healthy, whilst No. 13 presented us with a late condition in which the nervous elements had given way before the advance of the more lowly organised ones. Nor is it uninstructive to contrast these cases with those clinically normal ones, in which trivial changes were noted—changes however clearly tending in the same direction, though destined to remain in this very incipient stage; the changes indeed in these cases would very probably have remained in this incipient condition however long the patients had lived, though some might perhaps have advanced had sufficient time been allowed them.

In cases of primary atrophy such as No. 28, the pathological condition reached is eventually substantially similar, though here perhaps the sequence of changes may possibly be reversed at the distal extremity of the nerve.

As before mentioned, in the appended table details are given of all the cases examined. We do not think that there is any point in this, which calls for explanation, that has not before been commented on. As regards the refraction it will be seen, that there were eight cases of Hypermetropia and one of Hypermetropic Astigmatism; seven of Myopia, and two of Myopic Astigmatism; whilst the remaining 48 were either emmetropic, or the state of the refraction has not been recorded, in which latter case, the patients were in all probability emmetropic.

Though not strictly coming under the head of conditions of the fundus oculi, we have added a column for the state of the pupils, which have been recorded in nearly all the cases; we do so in order to emphasise the fact—not, we think, sufficiently known in this country—that there is a great tendency for the pupils in general paralysis to be inactive to light, whilst retaining their normal movement with accommodation—that is, to approach the Argyll-Robertson pupil, so common in locomotor ataxy. There is, however, as will be seen, much variation in this respect.[1]

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[1] For a paper on the condition of the pupils, &c., in "General Paralysis," &c., see Bevan Lewis, in vol. iii. of Ophthalmological Society's Transactions.

Mania.—Taking all varieties of mania together, we have examined 82 cases: 6 of these cases were examined twice, and 1 three times, making a total of 90 separate examinations. Fifteen of these cases were males, and 67, females; the average age of the patients was 37·81 years.

Of these 82 cases, in 70 (85·36 per cent.) the optic discs and main retinal vessels were perfectly normal, whilst in 12 (14·63 per cent.) the condition of these parts was either abnormal or doubtful; that is, in these latter (doubtful) cases, the changes were so slight that no decided opinion as to their abnormality could be formed.

Taking the cases more in detail, we find that out of 34 described as "acute," 30 were normal, and 4 abnormal, or doubtful; out of 7 cases of "puerperal" mania, 2 were normal, and 5 abnormal or doubtful; whilst out of 26 cases of chronic mania, only 2 were described as abnormal or doubtful, and out of the 15 cases of the delusional form of mania only one, all the others being perfectly normal.

We append a table showing the condition of the optic discs, &c.; in these 12 abnormal or doubtful cases, upon which a few remarks may be made. (1) The changes are exceedingly trivial or inconsequential.

In No. 1, the discs were almost certainly normal; in No. 3, an old syphilitic choroiditis accounted for the majority of the changes noted; in No. 5, the only abnormality was that the veins were too full, but as in a subsequent examination this undue fulness had subsided, we have thought it advisable to include the case; in No. 10, the high degree of myopia was responsible for almost all the changes noted, though the appearance of atrophy in the right disc may have been independent. We have preferred to give these cases in full, in order that the reader may form his own conclusions upon them, but we are decidedly of opinion that some at least of the cases are normal.

2. It is, perhaps, worthy of note that almost all the changes noted, such as they are, are in the direction of abnormal redness of the disc; but in this connection we must not lose sight of the fact, that it is often as difficult to tell where the physiological limit of tint ends and the pathological begins, in the case of redness as in that of pallor.

3. The table brings out clearly the numerical preponderance of changes in the acute cases over the chronic.

Some further remarks are requisite with reference mainly to some of the normal cases. One of the cases was examined twice in the course of the acute attack, and another three times, but the discs presented precisely the same appearances at each examination.

We were able to secure a good view of the discs in 3 cases *during an acute maniacal paroxysm;* in one of these cases the discs were thought to be too red, and this case is included under the abnormal ones in the table (No. 9); in the 2 other cases the discs were noted to be perfectly normal; in one of these, however, it was only possible to make an indirect examination, whilst in the other the left disc only was examined, but a thoroughly good view of it was obtained by the direct method.

We have examined 3 cases shortly (1 to 3 or 4 days) after the subsidence of a maniacal paroxysm; in these the discs presented perfectly normal characters, no abnormal redness or pink suffusion being noted.

In 4 cases the condition of the discs was noted in the same patient, during the maniacal attack, and after convalescence; in one of these, which is included in the table of abnormal cases (No. 5), an undue fulness of veins was noted during the attack, which was not present when the patient was convalescent; in the 3 other cases the discs presented precisely the same appearances at each examination.

In a few of the 70 cases in which the optic discs and vessels were normal, some changes in the choroid were found. In one of these there was evidence of old choroiditis; in one, large posterior staphylomata, and in two others, small ones, due to the presence of myopia; whilst in one other small crescents were noted without any myopia; in another case again there was slight choroidal atrophy all round the discs, and in 3 others a little pigmentary disturbance of the choroid around or on one side.

In one case there was a detachment of the retina in the right eye. We have thought it well just to mention these points, but consider they have no bearing upon the subject of our paper, for though we have included one or two such in our table of abnormal cases, this has been simply because the changes in the choroid were here associated with others in the discs, which latter were possibly independent of the former.

Melancholia.—In all, 49 melancholiacs have been examined, 8 of these were males, and 41 females. The average age of the patients was 38·93 years. In 40 of these cases (81·63 per cent.) the optic discs and main vessels were absolutely normal, whilst in the remaining 9 cases (18·36 per cent.) slight changes were found. More in detail; out of 7 cases of active recent melancholia, 5 were normal, and 2 abnormal or doubtful; out of 32 cases of the passive recent form, 27 were normal, and 5 abnormal or doubtful; whilst out of 10 cases of the passive variety which had become chronic, only 2 were in the latter category.

We consider that some of the changes noted are of very doubtful abnormality, but we have preferred (as in the case of mania) to give the cases in detail, in order that any one may be able to form his own opinions on the subject.

We may, however, remark concerning some of the cases in the table, that No. 2 was very probably quite normal; in No. 4, the only abnormality noted was that the vessels were rather small; in No. 3, the appearances of atrophy were most probably due to the patient's alcoholic excesses, which were almost certainly responsible for her insanity; whilst in No. 6, the changes noted were due to the presence of chronic Bright's disease.

We may just add, that in the 40 normal cases there were 2 in which choroidal atrophy was present round the discs, in one of which it took the form of a small crescent; in 2 other cases there were pigmentary deposits round the discs; whilst in yet 2 others a few small white spots were noted in the retina, due in one of these cases to chronic Bright's disease.

Mental stupor.—Two cases of mental stupor were examined, both during the attack and after convalescence; the condition of the discs is given in full in Table IV.; it would seem from the descriptions that the discs were perhaps slightly more pale during the condition of stupor than when the patients were convalescent, but the difference was so slight that we think it would not be safe to draw any conclusions.

Dementia.—We have examined in all 61 cases of dementia, 7 of these were males, and 54 females; the average age of the patients was 46·80 years. Of these 61 cases, in 46 (75·40 per cent.) the optic discs, &c., were perfectly normal; whilst in the remaining 15 (24·59 per cent.) these parts presented some degree—either slight or considerable—of departure from the normal, the changes in some cases being so slight that no decided opinion as to their abnormality could be formed.

Examining the cases more in detail we find that out of 41 cases of dementia, secondary to mania, 33 were normal, and 8 were abnormal or doubtful; whilst out of 8 cases of primary dementia, 5 were in the former, and 3 in the latter category; of organic dementia again, out of 8 cases, 6 were normal, and 2 abnormal or doubtful; only 4 cases of senile dementia were examined, 2 of which were normal, and 2 the reverse.

As in previous cases, we append a table giving in detail the conditions noted in the 15 abnormal or doubtful cases.

It will be observed that in the majority of the cases given in the table, the changes were in the direction of abnormal redness; but the same remark applies here that we made in the case of mania, as to the difficulty there often is in defining the physiological limit of colour, and probably some of the cases included in the table were normal. In Nos. 7 and 12, the changes described were doubtless altogether due to the Bright's disease from which the patients suffered.

In 3 of the 46 normal cases, considerable posterior staphylomata were present, due to myopia; whilst in one other there was a small crescent without any abnormality of refraction being detected. In another case there was an irregular patch of choroiditis round each disc, whilst in 3 others there was more or less pigmentary disturbance of the choroid in this situation.

Epilepsy.—Of insanity associated with epilepsy we have examined 48 cases; 8 of these were males, and 40 females; the average age of the patients was 33·20 years.

Taking all the 48 cases together, in 41 (85·41 per cent.) the optic discs and main retinal vessels were perfectly normal; whilst in 7 (14·58 per cent.) these parts were either abnormal or doubtful.

Of epilepsy associated with imbecility 20 cases were examined, 15 of which were normal, and 5 abnormal or doubtful. Of epilepsy, with slight impairment of the mental faculties, 20 cases were examined, only one of which showed any departure from the normal, and that very slight; whilst of the cases of epilepsy combined with considerable dementia, only one case out of 8 showed any tendency to abnormality, and here again the change was very trivial. As in other cases we append a table, showing the condition of the optic discs in the 7 abnormal (or doubtful) cases, from which it will be seen that the changes, such as they are, are very slight, and are chiefly in the direction of abnormal (or doubtfully abnormal) redness. Choroidal disturbances were noted in 5 of the 41 normal cases; 2 of these were merely myopic crescents; in one there was a zone of choroidal atrophy round the discs, and in one other some slight progressive choroiditis in the same situation, associated with chronic Bright's disease; while in yet another case there was slight pigmentary disturbance of the choroid around the discs.

It is perhaps worth a note, that out of the 48 cases no fewer than 20 presented errors of refraction; of these 12 were hypermetropic, and 2 had hypermetropic astigmatism; 2 were myopic, and the other 4 had myopic astigmatism.

A few observations were made with reference to the condition of the discs and retinal circulation before and after the occurrence of fits.

In one case a fit came on whilst the disc was under observation, but it was unfortunately impossible to keep this in view during the progress of the attack; immediately afterwards, however, the disc was thought to present a distinctly pink tinge, as compared with its condition just prior to the attack, when it was very pale.

One case was examined some hours after a series of 35 fits, but no abnormality was detected. In another case, a good view was obtained by the direct method immediately after a fit, which was one of a series of status epilepticus, comprising something like 60; the disc was normal, of a red tint, with well-defined edges; the veins were full, but could not be described as abnormally distended; these characters remained constant during an observation of about 20 minutes.

We may mention in this connection, that in the case of a general paralytic (No. 33 in Table 1), who died after a series of convulsions, a view of the left disc was obtained by the indirect method just as a severe fit was passing off; the veins then appeared distinctly distended; a good view was subsequently obtained by the direct method and maintained for some minutes; the discs and vessels then appeared normal, though the blood in one of the main veins was perhaps a little darker than usual.

Imbecility.—Five cases of imbecility have been examined, *not* complicated with epilepsy; 3 of these were males, and 2 females; the average age of the patients was 22-80 years.

The discs were perfectly normal in every case.

Taking again the whole series of cases examined, one or two further remarks have to be made.

A point of some little interest which forced itself upon our attention, was the number of cataracts we came across; including all cases (slight and considerable) of opacities and striæ in the lenses, we noted that 43 out of the 313 cases were so affected—a proportion of 13·73 per cent. The average age of the patients was 54·72 years. We are not acquainted with any statistics bearing on this subject, but we should think that this was a much higher percentage than occurs in the population at large of the same average age. If this supposition be well founded, it would furnish another interesting exemplification of the general impairment of nutrition, so common in the insane.

We think it just worth mentioning, that out of the 313 cases we met with 4 in which the condition known as "opaque nerve fibres" existed in connection with the disc; the extent to which this occurred varied considerably in the 4 cases, and was in none of them very extensive; the condition, as is well known, has no pathological significance.

It is also perhaps worth a note, that in not a few cases we met with tortuosity of the vessels, this condition being extreme in the person of one man suffering from the delusional form of insanity. We mention this as some observers appear to have attached weight to the circumstance; but in our own cases we feel confident that the condition was purely physiological, our experience furnishing us with numerous similar cases, in persons other than lunatics, in which it was not legitimate to invoke a pathological origin.

Only two cases of retinitis pigmentosa were met with, and both these occurred in the persons of female General Paralytics, and have already been referred to; in one case the changes were confined to one eye.

It would be foreign to the purpose of this communication to examine in detail all the previously published observations on this subject, even had we the material before us to enable us to do so. In Dr. Gowers' valuable work on the ophthalmoscope, references are given to some of the Continental publications on the subject, and the striking discrepancies between the observations of different observers is commented on. We will content ourselves therefore with some remarks on the observations which have been published in this country.

First then, as regards General Paralysis.

To Dr. Clifford Allbutt belongs the credit of being the first —in this country at least—to break ground on the subject, and his observations, first published in the 51st volume of the Medico-Chirurgical Transactions, are now to be found incorporated with his work on the ophthalmoscope.

Of 53 cases of General Paralysis examined, Dr. Allbutt only found 5 that were healthy; of the remaining 48 he found "atrophy of the optic discs in its various stages in 41 cases," and 7 cases were marked as doubtful; and the conclusion drawn was "that atrophy of the optic nerves takes place in almost every case of General Paralysis."

This conclusion is not in accordance with the results of our own observations.

On going carefully through the cases given in detail in Dr. Allbutt's tables, we find amongst the 41 cases, 4, in which pure hyperæmia was present, without the slightest appearance of atrophy whilst the case was under observation; in 14 others, some amount of atrophy was combined with the appearances of congestion, or these latter had been noted at some previous examination; the remainder must therefore be intended for cases of pure atrophy. After a careful perusal of the descriptions however, we are unable to reconcile some of these with the conclusions drawn from them; at the least, we should say that some of them were of very doubtful abnormality. We note also that three cases of "Amaurosis" are given, without any mention being made of the appearance of the discs, which we therefore presume were not examined; we cannot, however, consider that to include these cases under the head of "atrophy" is altogether satisfactory, seeing how many misleading factors come into play in a demented General Paralytic, in the third stage of the disease.

We may also mention that in the case numbered 31 in Dr. Allbutt's tables, the atrophy of the left disc is at least as likely to have been due to the syphilitic choroiditis with which it was associated, as to have had an independent origin. Making every allowance for doubtful cases however, the percentage of abnormal cases in Dr. Allbutt's tables is very much higher than that which we have ourselves experienced. As regards the nature of the lesion however, in the abnormal cases, we are quite in harmony with Dr. Allbutt, the changes noted by us being clearly practically identical with those so well described by that observer.

In Vol. II. of the West Riding Asylum Reports (1872), will be found a paper by Mr. Aldridge, entitled "Ophthalmoscopic Observations in General Paralysis." Mr. Aldridge examined 43 cases, the details of which are given in his tables. Though the author does not distinctly state that all his cases were abnormal, he leaves it to be inferred that they were so; for on p. 225 of the work referred to, he states that "only two conditions of the optic nerve were observed," and then goes on to describe these conditions, as those of neuritis and atrophy; whilst on p. 227 he distinctly states that atrophy was present in greater or less degree in all the female patients (13 in number) that he examined.

Here again we note agreement as regards the nature of the change with the prior observations of Dr. Allbutt and those now published by ourselves. The numerical proportion of abnormalities, however, detected by Mr. Aldridge, being so greatly in excess of anything we have observed, we cannot resist the suspicion that some error has crept in to vitiate his conclusions; nor indeed is internal evidence of this wanting; for on p. 226 of the work before us, we read: "The disease generally ends by one side of the disc, usually the inner, becoming white and atrophic, whilst the inflammatory changes are still in progress at the other. *In some cases again another course seems to have been taken, for in them we find that the disc has a white rim, and that a very large and shallow excavation of an extremely pearly white tint occupies the centre, the remaining portion being said to be of a greyish-pink tinge.*"

The italics are our own, and we emphasise this passage because we recognize in it a good description of a perfectly normal disc, with well-marked physiological cup.

We cannot indeed resist the conclusion, that this physiological cup (which, as is well known, varies greatly in size) has had to do duty for atrophy in not a few cases.

Space will not permit of a critical analysis of all the cases, but we may mention Nos. 25, (in which "atrophic changes were noticed," p. 228) 26, 27, 29, and 32, as instances in point: thus, of No. 29 it is stated: "Right eye: O.D., well defined by a narrow white rim of medium red tint, and having central bright white spot. Left eye: O.D., well-defined, very narrow white rim, it also has a small central white spot, the rest being of a medium pink glow."

As regards the main vessels, Mr. Aldridge leaves it to be inferred from his descriptions that in not a single case were these of normal calibre.

On the subject of General Paralysis, Dr. Gowers says,[1] "Most of the cases I have examined in various stages of the disease, presented perfectly normal conditions. In one case only was there the appearance of simple congestion of the disc."

Nettleship (quoted by Gowers) has recorded[2] an interesting case, in which optic nerve atrophy preceded the symptoms of General Paralysis.

[1] 'Medical Ophthalmoscopy,' 2nd edition, p. 178.

[2] 'Ophthalmic Hospital Reports,' vol. ix., p. 178.

A more recent observer, Dr. Lawford, has described (Transactions of Ophthalmological Society, vol. iii. p. 221), 7 cases of optic nerve atrophy in General Paralysis; it is not, however, stated from what proportion of cases these results were obtained, but only that 3 of the number were found in an examination of 22 cases of the disease.

It is interesting to note, in connection with the question of a relation between optic nerve atrophy and spinal symptoms in General Paralysis, that 5 out of these 7 cases had spinal symptoms, one posterior sclerotic, and 4 lateral sclerotic.

As regards insanity other than that associated with General Paralysis, the most important observations made in this country, so far as we are aware, are those by Dr. Clifford Allbutt, which are to be found in his work on the ophthalmoscope. Speaking broadly, we may say that Dr. Allbutt found a much larger proportion of changes than we have ourselves noted. Space will not permit of our analysing the cases in detail, nor indeed is it necessary to do this, as Mania is the only form of insanity with which an attempt has been made by the author to associate a definite change in the fundus oculi. We will briefly examine the main conclusions drawn in this case. Dr. Allbutt states, "that both in mania depending upon organic causes and in functional mania, the back of the eye if observed within a few days after a paroxysm, presents a vascular suffusion or pinkiness, so great after severe paroxysms as to obscure the discs." We regret that our attention was not forcibly directed to this statement at a sufficiently early period of our investigation to have examined it with the attention it deserves; so far, however, as our cases go they do not support the conclusion, for by a reference to what we have written under the head of Mania, it will be seen that we have examined three cases with reference to this point, in none of which was any abnormality detected. Dr. Allbutt further states "that during the paroxysm, on the contrary, the disc is anæmic, perhaps from spasm of the vessels;" this conclusion is, however, founded upon one observation only, and our own cases do not support it, for it will be remembered that we examined three cases during an acute maniacal paroxysm, in two of which the discs were perfectly normal, whilst in the third the changes noted were in the direction of abnormal redness, rather than of abnormal pallor.

In Acute Dementia, Mr. Aldridge found "a state of retinal anæmia,"[1] and adds "the optic discs are pale." In most of our own cases the discs were normal; in one only did we note "tint inclining to pale, arteries rather small;" whilst in another case the discs were "very red." (See Table V., Nos. 9 and 10.)

In Epilepsy Mr. Aldridge states,[2] as the result of an examination of 102 cases, that "in a great majority of them a condition of hyperæmia of the retina and optic discs was found." As will have been seen, our own observations gave altogether different results.

Conclusions.—In all questions dealing with the condition of the fundus oculi in insane patients, it is necessary to draw a clear line of distinction between the disease known as General Paralysis of the Insane, and other forms of insanity. With this proviso we venture to draw the following conclusions:

1. That in Insanity proper (including all forms other than General Paralysis) changes in the fundus oculi are found in a small minority of cases; but that when allowance is made for changes depending upon associated constitutional conditions, errors of refraction, &c., the number of cases in which a connection between the mental (cerebral) state, and the accompanying change in the fundus oculi, can be so much as suspected, is very small.

As a corollary from this we may say,

2. That in Insanity proper no connection can be traced between the condition of the fundus oculi and the patient's mental state.

3. That in the majority of cases of "General Paralysis of the Insane," the fundus oculi presents a perfectly healthy appearance.

4. That in a minority of cases clear and precise lesions are found.

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[1] 'Ophthalmoscopic Observations in Acute Dementia,' West Riding Asylum Reports, vol. iv. (1874), p. 296.

[2] 'The Ophthalmoscope in Mental and Cerebral Disease,' West Riding Asylum Reports, vol. i., p. 87.

5. That these lesions fall into two main classes, the one tending in the direction of slight neuritis, the other in that of atrophy.

6. That in the former class the affection declares itself as a hyperæmia of the discs, the edges being softened and indistinct, so that in some cases they can be traced with difficulty or not at all; and that these conditions tend—if the patient live long enough—to be replaced by atrophy, so that at length complete disorganisation of the nerve may take place. The changes are essentially chronic in their course.

7. That though atrophy of the optic nerves may thus succeed to a slight chronic interstitial neuritis, it is also not unfrequently primary *at the disc;* the atrophy may be complete, the patient becoming quite blind.

8. That the pathological basis underlying the appearances of slight neuritis, may be broadly characterised as a tendency to overgrowth in the connective elements of the nerve; the trabeculæ not only getting greatly hypertrophied, but the neuroglia corpuscles also becoming very large and numerous; these parts thus grow at the expense of the nervous elements, which subsequently atrophy.

9. That in the cases of primary atrophy, the pathological appearances eventually reached, though somewhat similar, may possibly take place in the reverse order *at the disc*; the nerve fibres being the first to dwindle, and the fibrous elements—trabeculæ &c., subsequently taking on increased growth.

10. That in a considerable proportion of the cases in which atrophy of the optic discs is met with, spinal symptoms are prominent in the disease, these symptoms pointing in the direction of posterior or lateral sclerosis of the cord; but that this connection is by no means invariable.

ON ALCOHOLIC PARALYSIS.

BY J. DRESCHFELD, M.D., F.R.C.P.,

Professor of Pathology, Victoria University; Physician, Royal Infirmary, Manchester.

The effects of alcohol on the nervous system manifest themselves in various ways, chiefly according to the particular part of the nervous sphere attacked.

Whilst in many forms of chronic alcoholism the brain seems to be the chief organ to suffer, there is one affection, alcoholic paralysis or alcoholic paraplegia, in which cerebral symptoms are either altogether absent, or play a mere subordinate part, while most of the symptoms are referable to the spinal cord or to the peripheral nerves. The symptomatology of this affection has been well described in this country by Wilks ('Diseases of the Nervous System'), Lockhart Clarke ('Lancet,' 1872), Reginald Thompson, ('Med. Chir. Trans.' 1868), and more recently by Glynn, ('Liverpool Medico-Chir. Journal'), and Broadbent ('Lancet,' Feb. 16, 1884). On the Continent, the principal workers at this subject have been Leudet ('Arch. Gén. de Méd.' 1867), Lancereaux ('Gaz. Hebd.' 1865 and 1881), Moëli (Berl. Klin. Wochenschr. No. 14, 1884), and Charcot (see article by Féré in 'Prog. Méd.' June 14, 1884).

Our knowledge of the pathological anatomy of this affection is, however, still meagre, although the observations of Lancereaux, Moëli, and Broadbent, tend to show that the lesions concern chiefly, if not entirely, the peripheral nerves. As a small contribution to this interesting subject, I may be permitted to describe the lesions found in one case which was recently under my care, and to allude to a few cases which I have from time to time observed.

The disease runs by no means the same course in every case, nor does it invariably present the same grouping of symptoms, though certain of these symptoms are always, or almost always, present. Considering, however, the more prominent characteristics, we may, I think, divide alcoholic paralysis into certain types.

The first may be fitly designated *Alcoholic Ataxia.* Of this form I have seen several cases, and other observers have also alluded to it; thus Wilks ('Dis. of the Nervous System,' p. 225) mentions the case of a young woman "whose symptoms very much resembled those of locomotor ataxy, both in the character of the pains, which were like electric shocks, and also in the mode of progression." Leudet (loc. cit.) alludes to the difficulty of distinguishing such cases from true ataxia, and Lancereaux and Charcot also mention the ataxic symptoms. The cases of this description which I have seen were men, from 30 to 40 years old; the symptoms had come on more or less gradually, and consisted chiefly of marked inco-ordination in the lower extremities, absence of tendon-reflex, and lancinating pains. There was neither atrophy nor paralysis.

A patient whom I saw recently with Dr. Roe, of Patricroft, and who had been greatly addicted to alcohol for years, had already had one attack of paralysis, from which he completely recovered, after giving up alcohol. Having again fallen into bad habits, the old symptoms recurred (inco-ordination, lancinating pain, absence of tendon-reflex and slight loss of control over the bladder at night). Dr. Roe informs me that the patient's habits are still very intemperate, that his ataxic symptoms have remained unaltered, but that the patient is now suffering from rapidly advancing phthisis.

A case of very similar description I saw with my colleague, Mr. Jones: here also the symptoms came on after a series of alcoholic excesses, and I am informed have now quite subsided. The patient's family history was free from any neurotic taint, and he had not had syphilis at the time I saw him, though he has contracted it since then.

Another case I had an opportunity of watching for some time in the out-patients' department of the Manchester

Infirmary. A man, æt. 35, an excessive drinker, suffered from the symptoms described above, which came on in the course of three weeks, but subsided completely in three months. The recovery in his case was complete, the tendon-reflex being again established some months after all the other symptoms had disappeared.

These cases differ from those of the second type to be described below, in the absence of hyperæsthesia, hyperalgesia, vaso-motor disturbances, paralysis, atrophies and mental disturbances. The individuals attacked are chiefly men, and the symptoms disappear with the discontinuance of alcohol. In the diagnosis of such cases of pure alcoholic ataxia, one has of course to bear in mind how far alcohol may simply play the part of an etiological factor, and how far alcoholism and ataxia may be mere coincidences; moreover in the ordinary cases of ataxia, the symptoms, especially if they come on somewhat acutely, often show for a time a marked improvement. In the above cases, however, the ataxia followed so soon after the excesses, and so completely disappeared, that I have no hesitation in pronouncing them as alcoholic. This view is still further supported by the fact, that symptoms of ataxia are again seen in the second type to be described (especially the absence of tendon-reflex and the lancinating pains); moreover, some of the symptoms of ordinary locomotor ataxy, such as the arthropathies, the oculo-motor pareses, spinal myosis, &c., symptoms which we may consider spinal, are absent in the alcoholic ataxia. The symptoms observed harmonise well with the pathological anatomy which underlies the alcoholic paraplegia,—for as the changes observed chiefly concern peripheral nerves, we can understand the symptoms if we suppose that in the ataxic type we have an affection, a multiple neuritis, especially of the sensory nerves.

The second type of *alcoholic paralysis*, which deserves better the name of *paralysis*, is far more common, and is that which most observers have described.

This affection chiefly attacks females, who, though they deny the abuse of alcohol, have suffered from symptoms of chronic alcoholism (morning vomiting, chronic gastric catarrh, hæmatemesis, &c.), previous to the appearance of the nervous troubles. These commence more or less acutely, and consist of sensory, motor, vaso-motor, and trophic disturbances.

The sensory disturbances are, as a rule, well marked and very characteristic, and consist of extreme hyperæsthesia of the feet and other parts of the lower extremities, with or without hyperalgesia; perversion of the sense for temperature (all objects when brought in contact with the skin feel cold); there are often present, in addition, lancinating pains in the lower extremities, and pain when pressure is applied along the vertebral column. The hyperæsthesia is often followed by anæsthesia or retarded sensibility, the hyperalgesia by analgesia.

The motor troubles consist of more or less marked paralysis, attacking the lower, and sometimes the upper extremities; in many cases the extensor muscles are chiefly affected, while in not a few cases the paralysis is more general. The superficial and mechanical reflexes are often diminished, the tendon-reflexes are nearly always absent; contractures have only been noticed by a few observers (Wilks, Buzzard). The vaso-motor affections consist chiefly of redness of the feet or hands, occasionally also of other parts of the body, and œdema, especially seen at the ankle, the dorsum of the foot, and the back of the hand. The nerves and muscles show marked degenerative reaction, though we have but few detailed statements on this subject.

Cerebral symptoms, such as insomnia, restlessness, more or less delirium and hebetude, though absent at first, often make their appearance during the course of the disease, and are frequently the cause of death, unless other complications should occur, such as phthisis, fatty degeneration of liver, &c.

Disorders of the circulatory, digestive and respiratory organs, are often seen during the progress of the disease, but are rather complications, or the primary effects of alcohol on these organs, than secondary manifestations of the nervous affection under consideration.

The prognosis varies very much. Many cases recover when they come early under the care of the physician, and when uncomplicated; some recover for a time, and afterwards relapse; whilst in others the disease pursues a steady downward course, death ensuing from the cerebral symptoms, from exhaustion, or from some intercurrent affection, as is so often seen in other forms of chronic alcoholism.

The following case which was recently under my care, and which I was able to follow to its fatal issue, may be taken as a fairly typical example of this form of alcoholic paralysis, though it differs from some of the reported cases in the extent of the paralysis, which affected the lower extremities more uniformly than is ordinarily described.

The notes of the case were taken by one of my clinical clerks, Mr. E. E. Bowden.

Mary P., æt. 49, married, was admitted into the Infirmary on April 1, 1884.

Previous History.—The patient has resided in Manchester for 30 years; has had six children, all of whom have died, and has had one miscarriage. She has worked in a laundry, and admits having taken stimulants (though the patient denied any excesses, inquiries made of her neighbours satisfied us that she had been a chronic alcoholic; moreover, in her delirium she continually called out for beer). She has been in somewhat straitened circumstances, but enjoyed very good health up to ten years. She had small-pox some years ago, but has never had any other illness; the catamenia ceased two years ago, before which time they were very profuse.

History of Attack.—Two years ago the patient began to feel weak, especially in her knees, and she staggered then slightly in her walk; she also noticed that in dressing, when putting her garments over her head, she had to steady herself; she gradually grew weaker, and during the last few weeks before admission the weakness has so much increased, that she is now unable to walk.

State on Admission.—The patient lies on her back, and is unable to rise or turn over without great difficulty; her face has a haggard, almost demented expression; she is somewhat emaciated, and has a large bed-sore on her back.

The patient is fairly intelligent, and complains much of insomnia and slight headache; there are no other cerebral symptoms.

There is a considerable amount of paresis of the upper and lower extremities, and also of the muscles of the back. The patient is scarcely able to walk or stand by herself, and with her eyes shut would fall immediately. She lies with her legs flexed, and extends them with great difficulty; can flex and extend her foot; can abduct and adduct the legs, but does so with difficulty and pain. The grasp of her hands is weak; can flex and extend her wrists and elbow; the effort of rising in bed causes great pain. There is great pain in both lower extremities, and patient shrieks when the legs are touched; the left knee is specially painful, the skin over it is reddened, and the joint is slightly swollen; there is also great pain in the back and loins, and along the whole of the vertebral column; there are no spots of either anæsthesia or analgesia; all the muscles seem equally atrophied and flabby; the superficial reflexes are fairly normal; the tendon-reflexes are absent; there is some diminution of the muscular sense in the upper and lower extremities.

The pupils are contracted, react very little to accommodation, and not at all to light. The tongue is furred and glazed; there is loss of appetite; the patient suffers from thirst and diarrhœa; the thoracic organs are found normal on physical examination; there is no cough and no expectoration; the liver and spleen appear normal on percussion; there is retention of urine, and the catheter has to be used; the urine is alkaline, has a sp. gr. of 1011, contains a large quantity of pus and a considerable amount of albumen.

The heart is found normal; the pulse regular, but quick, feeble, and easily compressible.

The temperature was in the morning 99, in the evening 100·4.

April 3rd.—The patient's condition is much the same as on admission.

Faradic Contractility.—Direct application to muscles; no contraction of the dorsal flexors of the right foot, even with the strongest current; those of the left foot react slightly to strong current.

Quadriceps femoris does not respond to any current.

Hamstring muscles and biceps react slightly.

Flexors and extensors of fingers react but slightly with the strongest current.

Biceps and triceps react somewhat better.
Erb's point scarcely any reaction.
Galvanic reactions.

LOWER EXTREMITIES.

	Kat. Cl.	ACl.	A.O.
Left Ext. brev. Dig. . . .	45	40	..
„ Exten. Prop. pol. „ Tib. Antic.	45	40	45
Right Tib. Antic. „ Exten. Prop. pol.	45	40	..
Left Rect. fem.	45	40	..
Right „ „	50	40	..
Left Hamstring	50	35	..

UPPER EXTREMITIES.

	Kat. Cl.	ACl.	A.O.
Left Ext. digit.	30	20	20
„ Triceps	20 (slight)	20	25
		25 produces tetanus	
Right Ext. dig.	30 (slight)	25	25
„ Triceps	35	30	..
Left Flex. digit.	35 (slight)	20	35
„ Erb's point	40	45	..
Right Erb's point . . .	40	..	45

Diagnosis.—Alcoholic paralysis. This diagnosis was arrived at without any great difficulty. The history of the case pointed strongly to a chronic alcoholic intoxication, and the symptoms on admission were well marked and typical. One might have thought of ordinary ataxia; but the extreme sensibility to pain and touch, and the degenerative reaction of the muscles, were against such an assumption. Against sub-acute or chronic poliomyelitis the sensory symptoms told with equal force. Against chronic meningo-myelitis pointed the fact that the disease commenced without pain, that paresis and inco-ordination were the first symptoms observed, and that the tendon-reflexes were absent. Against paraplegia dolorosa, such as is observed in cancer of the vertebræ, pointed the absence of any tumor, the absence of cachexia, the chronicity of the case, and the painless implication of the upper extremities.

There remained, therefore, scarcely any other disease but multiple peripheral neuritis, and with the history before us, and the absence of any evidence showing that any other toxic agent, such as lead, arsenic, &c., was the cause of the symptoms (though such poisons would scarcely produce the sensory disturbances observed), we had no difficulty in arriving at the diagnosis stated.

Progress.—(I extract a few statements from short-hand notes.)

April 7.—The patient is much worse. She is delirious at night; her speech is slow and muttering; she lies with her legs flexed, and can only extend them with great difficulty.

April 9.—The patient is much worse; the delirium is now constant; the temperature 95; the extremities cold and cyanotic; pulse, very feeble, 80 per minute; the paralysis more extensive.

April 10.—Last night the patient became comatose, and died early this norning.

Autopsy, by Dr. Maguire, Pathologist to the Royal Infirmary.—Some fluid in left pleural cavity, a little fluid in pericardium; aortic valves showed some chronic thickening; the lungs, slightly emphysematous, contained a few caseous nodules in both apices. The liver enlarged, pale and soft. The kidneys showed several patches of caseation in the cortex; the pelvis full of pus; the capsule thickened, but peeled off easily, and showed the surface of the kidney affected by tubercular deposit. The bladder greatly inflamed, its inner surface covered with muco-pus, its walls thickened and infiltrated with tubercular deposit.

The brain was found normal, the pia slightly thickened; the dura mater showed numerous Pacchionian bodies.

The spinal cord and its membranes, when examined in the fresh condition, appeared perfectly normal.

Some peripheral nerves, which were reserved for microscopic examination, also appeared normal.

The muscles were pale, but otherwise normal.

After hardening the cord, I examined portions of it microscopically, without finding any changes: examined by Weigert's method (' Fortschritte der Med.' 1884), numerous small nerve fibres were found in Clarke's columns. The ganglion cells in all parts of the spinal cord were perfectly healthy.

Several posterior roots of the lumbar region were examined nd found perfectly healthy.

Of the peripheral nerves, there were examined the two sciatics, the musculo-spiral and the anterior crural. These nerves showed the characteristic changes described by Lancereaux in a very marked fashion. In the sciatic, longitudinal and transverse sections showed most markedly the breaking up of the myeline, and in many places the breaking up of the axis cylinder also. Longitudinal sections showed the segmental changes described by Gombault, and seen by me in a case of lead paralysis (see Dr. Moritz's paper in the 'Journal of Anat. and Phys.' 1881). The muscles showed no peculiar changes when microscopically examined. The post-mortem changes showed then a multiple peripheral neuritis, chronic tuberculosis of the lungs, and tuberculosis of the kidney and bladder of more recent date. The diagnosis of alcoholic paralysis was thus confirmed by the post-mortem appearances, which were exactly those described by Lancereaux and Moëli.

Though hyperæsthesia and hyperalgesia are prominent symptoms in this form of alcoholic paralysis, yet I think that both these symptoms may be occasionally absent, and the motor nerves may be principally affected. Thus, not long since, I observed the case of a man, æt. 38, who suffered from far-advanced alcoholic cirrhosis of the liver, and who in the course of a few days lost the use of his arms and legs. When admitted into the Infirmary, March 22nd, 1884, about three weeks after the onset of the paralysis, there was noticed, besides the ascites and enlarged spleen, marked atrophy and paralysis in the upper extremity, affecting chiefly the muscles supplied by the radial, the posterior interosseous and median nerves, and more pronounced on the left than on the right side. The back of the hand was œdematous. There was also observed some paralysis of the lower extremities, affecting chiefly the extensors of the toes and ankle. In the sensory system there was found a perversion of the sense for temperature, namely, the patient felt, as intensely cold, anything that touched his arms or his legs. (I find this symptom stated in several of the recorded cases of alcoholic paralysis.) There was absence of tendon and superficial reflexes.

The electric reactions of the muscles, carefully taken by Mr. A. Mumford, one of my clinical clerks, showed marked degenerative reaction. The special sense-organs were normal.

The cerebral symptoms consisted chiefly of sleeplessness.

The patient was in the hospital some time, without showing any improvement. He left, and died at home, and we had no opportunity of making an autopsy.

The nervous symptoms in this case resembled more those seen in chronic poliomyelitis; yet the history, the onset, the perversion of the sense for temperature, the marked degenerative electric reaction and the associated cerebral symptoms (though these might have been due to the cirrhosis), induced me to look upon this case as one of peripheral multiple neuritis, affecting chiefly the motor nerves and caused by alcohol. The combination of cirrhosis with alcoholic paralysis has already been noticed by Lancereaux ('Gaz. Hebd.' 1865).

The *Anatomical lesions* then which underlie alcoholic paralysis, to judge from the post-mortem results of Lancereaux and Moëli, and those given in the case above, consist of a peripheral multiple progressive neuritis. Broadbent, in his case, found the cord normal, but was not allowed to examine any other part of the body. In one of Lancereaux's older observations the antero-lateral columns of the cord were found slightly sclerosed; in all the later observations the cord was found healthy.

The post-mortem lesions explain the symptoms very well, and also account for the diversity which we observe, according as the one or the other set of nerves are chiefly involved.

As regards *differential diagnosis*, the disease resembles in many respects the progressive multiple neuritis described by Leyden, Strümpell, Pierson and others, of which indeed it forms simply a particular species. It differs, however, from this non-alcoholic multiple neuritis in several particulars; it runs a less acute course, is less progressive, the paralysis is more confined to the extensor groups of muscles, and though there is a considerable amount of pain in the non-alcoholic form, there is not usually the very characteristic hyperæsthesia and hyperalgesia described above; the cerebral symptoms also are absent in the non-alcoholic form.

It further resembles other intoxications, notably those produced by lead.[1] Lancereaux has long ago drawn attention to this, and, pathologically considered, the lesions are the same in both; just as in alcoholic paralysis, so in lead paralysis, we have to do chiefly with peripheral nerve changes; though lead seems to select the motor nerves *par excellence.* The similarity of the action of lead and alcohol is further evident when we consider other diseases in which both play the important etiological element, as in gout, in the true cirrhotic kidney, and, I may perhaps also add, chronic endocarditis and some brain affections.

The *treatment* of alcoholic paralysis, which is often successful in the less advanced and uncomplicated cases, must have for its prime object the withdrawal of the alcoholic stimulant. Of other remedies, the opiates, cinchona (recommended by Leudet and Wilks), strychnia, hydropathy and the application of electricity seem indicated.

The iodide of potassium, which I have tried in some cases, has not given me any very encouraging results.

I wish now to draw attention to certain *visceral neuralgias* which, either alone or in combination with other nervous symptoms, are found in chronic alcoholism, and which I believe are caused by the alcohol. Thus, I was asked to see a lady who was an alcoholic, and who suffered from very intense colicky pains; these attacks would last for days; they were unaccompanied by vomiting or any other gastric disturbance. There was marked constipation. There was no reason to suppose lead-poisoning, nor did the gums show any blue line—yet iodide of potassium was freely given without benefit. Opium gave some temporary relief; the pains, however, recurred from time to time, and only disappeared when the patient was put under restraint, in her own home.

Another lady, aged 27, who was suspected to be an alcoholic, suffered from attacks which resembled very much angina pectoris; there was excessive cardiac dyspnœa; the pain radiated into the left arm, slightly also into the right, and there was also some epigastric pain. Physical examination of the chest-organs showed nothing abnormal, and beyond a very visibly pulsating abdominal aorta, nothing abnormal could be detected in the abdominal cavity. The cause of these symptoms was very doubtful to me; but when they disappeared, along with some other neuralgic troubles (chiefly affecting the trigeminus) after the patient was put under restraint in a home for dipsomaniacs, I strongly suspected that they had been due to alcohol; in this view I was confirmed, for the symptoms reappeared, when the patient left the home and recommenced her old habits. She was placed a second time into the home, and again improved; but died subsequently from pneumonia. I had no opportunity of making a post-mortem examination.

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[1] Atrophies produced by arsenic have been noticed by Gerhardt, 'Cbl. f. Klin. Med.' 1882.

These visceral neuralgias again resemble those produced by lead. Lead colic is common enough, and in a small epidemic of acute lead-poisoning produced by the inhalation of particles of lead-chromate, I have seen two cases, where, after the acute symptoms had passed off, symptoms very much like angina pectoris came on. These symptoms resemble most a vaso-motor disturbance, and as such we can easily understand their appearance in chronic alcoholism.

ON ATROPHY OF THE BRAIN IN IMBECILES.

BY FLETCHER BEACH, M.B., M.R.C.P.,

Medical Superintendent of the Darenth Asylum.

Many writers speak of two forms of atrophy of the brain, and it is a convenient method of describing the disease. In the first, there is incomplete development; in the second, there is loss of nervous elements which had previously been present. Taking first that variety in which there is incomplete development, and excluding that form of it in which the hemispheres are completely wanting and vegetative life merely exists, I would remark that cases illustrating this condition are commonly found in Asylums for Imbeciles. Microcephalic imbeciles are instances of this class. Microcephaly may be general or partial. Some portions of the brain may be too small or altogether absent. I have found the convolutions of certain parts very much reduced in size from non-development, while the remaining portions are too large or normal. The occipital lobes are usually arrested in growth in microcephalic cases, and the island of Reil may be left uncovered. The corpus callosum is sometimes shortened posteriorly, and cases are on record where this portion of the brain has been wanting and the commissures deficient.

As an instance of the arrest of growth of the occipital lobes, I would refer to a case related by Dr. Shuttleworth in the 'Journal of Mental Science for 1878,' in which the frontal and parietal lobes were comparatively well-developed, but the occipital lobes were quite rudimentary.

Usually, however, microcephaly is general, and the deficiency consists in the smallness of the hemispheres. In the 'Transactions of the International Medical Congress' for 1881 I have related two cases, in one of which the brain when removed from the body weighed only 7 ounces, yet a minute examination of it showed that nearly all the convolutions were present, though very small in size. In the other, the brain weighed 20½ ounces. Two brains of microcephalic cases have since come under my notice, and these weighed 20¼ and 24½ ounces respectively. The nerves of special sense are usually well-developed in microcephaly, and the ganglia of the base and the spinal cord are of nearly normal size. The cerebellum is relatively much larger than in the normal brain, often being in the proportion of 1 to 3 or 4, the ordinary relation being 1 to 8.

The following are illustrative cases:

G. L., aged 13 years, admitted into Darenth Asylum, January 19th, 1878. The father is a very hard drinker and often ill-uses the mother. He has an impediment in his speech, and so have all his side of the family. The mother is a temperate woman, but has a brother in Colney Hatch Asylum. Intemperance and hereditary predisposition to insanity are therefore present. The parents are not connected by consanguinity. G. L. had a small head when born. He did not walk till 5 years old, and has never spoken. The mother ascribes his condition to ill-usage by the father during pregnancy. He is a twin; the other child is dead, but his head was of normal size. There have been twelve children, of whom one, a girl aged 8 years, is also microcephalic. Seven have died, and of these one was microcephalic and died in convulsions.

On admission, G. L. was found to be rather thin, with a muddy complexion and a bird-like aspect. His head measured 17⅜ inches in circumference (3 to 4 inches below the normal size), 10⅞ inches transversely (3⅞ inches with calipers), and 11 inches antero-posteriorly (6 inches with calipers). Since his residence in the Asylum his head has increased a little in size, being now 18 6/8 inches in circumference, 11 inches in the transverse diameter, and 11⅛ inches in the longitudinal direction; but even now the outline of it, when compared with one of normal size, shows the great difference at once. His forehead was 3⅝ inches in breadth. He had good use of his limbs and was not subject to fits. In temper he was somewhat stubborn, but was easily managed. He could not speak, but could only make a sound like "ah." He was able to take his food without assistance, and could help in the ward, but he could not wash or dress himself without assistance. He had some power of observation, imitation, and attention, though but little memory. He went to school in the Asylum daily, and at the end of one year had learnt some letters of the manual alphabet, and could hold up one or two fingers to correspond with the objects shown to him. Two years afterwards he was able to read some words with the manual alphabet, could spell "cap," write "a, o, d" from memory, hold up fingers as far as four to correspond with objects, and could match colours and forms. He had also learnt to make himself very useful in household work, and could sew on buttons, and do simple repairs of clothing. Considering the size of his brain, I think we may say he has made fair improvement, though not so much as his sister, who has a smaller head.

A. P., admitted August 15th, 1877, was three years old on admission. Her parents are healthy, temperate, and not connected by consanguinity. A brother of the mother is subject to fits. The mother attributes the patient's condition to a fright which she had when four or five months advanced in pregnancy. This is the fifth child. The others are all healthy and have no mental defect. The child's head was small when born, and the mother thinks it has not increased much in size since, though the body has grown normally. She has never noticed passing objects, and has never been able to stand or hold anything. She is not subject to fits. Her thumbs have been drawn into the palms of her hands since birth.

A. P. when admitted was a fairly nourished child, of fair complexion, 30 inches in height. Her head measured 15¼ in. in circumference (5 to 6 inches below the normal size), 9½ in. transversely, and 10½ in. antero-posteriorly. As in the preceding case, the head has grown in size since admission, the corresponding measurements to those above now being 16, 9⅝ and 10⅝ inches. Her forehead is small and receding, tending to the oval in shape. In height it is 2 inches, and in width 3 inches. Her arms and legs are of normal size, the former measuring 11 inches, and the latter 13 inches in length; she cannot stand, though she can kick her legs about. She is subject to spasmodic retraction of the limbs, and though she can move her arms, she cannot hold anything. Her forearms are contracted on the arms and the fingers are clenched. Her legs are also contracted on her thighs, and cannot be straightened. She is placid in disposition and cheerful. Her habits are faulty. Her mental capacity is of course very small, but there is some power of observation and a little of attention. She has now been 4½ years in the Asylum, and during the whole of the time has been kept in the Infirmary, where she sits by day coiled up in a chair, and requiring to be fed, clothed, and generally attended to. She now seems to recognise her nurse, and laughs when pleased, but the improvement made is not great.

Both of these cases, and especially the latter, have heads much below the normal size, and correspondingly the brains are deficient in development. It is not, however, only the smallness of the quantity of the brain, but deficiency in quality which is involved. If we look at sections exhibited under the microscope, we shall see the great difference between a normal and a microcephalic brain. In the latter we notice the deficiency of cell processes, and the retraction of the protoplasm in the cell, accounting for the spaces so often visible. As to the causation of microcephalic imbecility, it has been held that it is due to the sutures of the skull closing in prematurely, and so preventing the growth of the brain; but, in opposition to this theory, there is the fact that many cases have been collected with open sutures. "Even in those cases where the sutures have closed in before birth, the question still remains whether the brain ceased to grow because the sutures are closed, or whether the sutures closed in because the brain ceased to grow; or lastly, whether both the brain and its coverings ceased to grow from a common cause."[1]

The second form of atrophy, viz., that in which there is loss of nervous elements which had previously existed, may present itself in various forms, but the most interesting is perhaps that in which there is atrophy of one side of the brain, usually the left, with coexistent atrophy of the limbs of the opposite side of the body. Now, though imbecility is not necessarily the result (vide cases quoted by Van der Kolk), yet examples of this disease are of fairly frequent occurrence in Asylums for Imbeciles. According to this author, everything depends upon the more or less healthy state of one hemisphere of the brain. "If, as from the nature of the case seldom occurs, the inflammation and affection of the pia mater has not extended to this hemisphere, if the grey matter under the cerebral convolutions has here continued perfectly sound, there is no reason why this remaining hemisphere should not be able to act without impediment in the exercise of those functions which are necessary to our mental powers, just as one eye sees as sharply though the other be lost. But where grey matter is injured in both hemispheres, particularly anteriorly, disturbance of the intellectual faculties will be inevitable."

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[1] Ireland on Idiocy and Imbecility.

An inflammation of the brain, meninges, or skull during fœtal life or early childhood, no doubt will cause the disease. The paralytic form of imbecility, for instance, seems to depend upon an atrophy of the brain caused by chronic meningitis or inflammatory processes in the cortical substance. Either of these causes may of course occur before birth or come on afterwards.

In the following case I believe atrophy of the brain to exist, and I think there is no doubt that it was produced before birth.

W. B., aged 9 years, was admitted into Darenth Asylum, November 14th, 1877. The parents were temperate and healthy people, and not connected by consanguinity. When the child was born, his right side was found to be powerless, in consequence, as is supposed, of the mother during pregnancy falling down and striking her side with great violence against a wall. On admission, his head was found to be flattened on the left side, but of fair size, being 19⅞ inches in circumference. He limped with his right foot in walking, and the fingers of his right hand were flexed in the palm. His mental capacity was fair. He could read a few letters, print with his left hand "a" and "o," count to 20, and knew a few colours. He has made good progress in school, so much so, that he is now able to read well from the 1st Standard, write and transcribe (with his left hand) in a book, work easy addition, subtraction and multiplication sums, and recognise all the colours and shades, and easy and difficult forms. He has progressed equally well in the tailor's shop, and, as a result of his industrial work, he is now able to straighten the fingers of his right hand, and make use of this hand in dressing himself and in his work.

The progress in such cases is nearly always fair mentally, though but little physically. In the one just quoted, greater physical improvement than usual has been made, but there are even now marked differences in length and size between the limbs of the healthy and those of the affected side.

RIGHT ARM (AFFECTED SIDE).

	Inches.
From point of acromion process to external condyle of humerus	10½
From external condyle of humerus to styloid process of radius	8⅛
From centre of wrist to end of middle finger	6⅝
Circumference round centre of deltoid	8
,, ,, ,, ,, ,, biceps	7½
,, ,, ,, forearm 1½ in. in front of inner bend of elbow	7½
,, ,, ,, forearm 1 in. in front of wrist	5⅝

LEFT ARM (UNAFFECTED SIDE).

From point of acromion process to external condyle of humerus	11¼
From external condyle of humerus to styloid process of radius	8⅜
From centre of wrist to end of middle finger	7⅜
Circumference round centre of deltoid	9½
,, ,, ,, ,, ,, biceps	9
,, ,, ,, forearm 1½ in. in front of inner bend of elbow	8¾
,, ,, ,, forearm 1 in. in front of wrist	6½

Every measurement of the affected arm is seen to be less than that of the unaffected one, and so with the leg.

RIGHT LEG (AFFECTED SIDE).

	Inches.
From inferior spinous process to external condyle of femur	15
From external condyle of femur to external malleolus	14
Length of sole of foot	9½
Circumference of thigh 10 in. below inferior spinous process	14½
,, ,, ,, calf 5 in. below external condyle	10⅝

Left Leg (Unaffected Side).

	Inches.
From inferior spinous process to external condyle of femur	16¼
From external condyle of femur to external malleolus	14½
Length of sole of foot	10
Circumference of thigh 10 in. below inferior spinous process	15⅜
„ „ „ calf 5 in. below external condyle	11¼

In the next case the date of the onset of the paralysis is doubtful. E. H., aged 18 years, was admitted into the Clapton Asylum, May 3rd, 1875, having been transferred from the Hampstead Asylum. I am indebted to Dr. Orange, Medical Superintendent of the Broadmoor Criminal Lunatic Asylum, for the following history. The father of E. H. had died in 1868, of cancer of the bladder. The mother was a patient in the Broadmoor Asylum, having killed one of her children while insane. The parents had been temperate people and were not connected by consanguinity. There was no family history of epilepsy or paralysis. This was the eldest child. There had been one other, who was killed by the mother, as mentioned above. E. H. was of sound mind at birth, and was doing well at Southall School, but was sent to Hampstead Asylum in consequence of epileptic fits, from which she suffered. These commenced at the age of 2½ years (cause unknown). Her intellect had become affected by the fits.

On admission, she was a fairly nourished girl of dark complexion, with loss of power of the right upper extremity (the forearm being contracted on the arm, the wrist on the forearm, and the fingers flexed in the palm of the hand), and weakness of the lower one on the same side. There was no means of ascertaining the cause of the paralysis, or when she was first afflicted with it. She was still having epileptic fits. She talked and answered questions with considerable intelligence, was of a quiet disposition, had pleasing manners and was a general favourite. Her mental capacity was very fair for an imbecile. She could read from the 3rd Standard, and write very well with her left hand. She had made considerable advance in arithmetic, being able to do simple and compound reduction sums. She was fond of music, and knew all the colours. From this it will be seen that she was an imbecile of a very high type. She made progress at school in the intervals between the fits, and was able to sing at the entertainments. In October of the same year she was noticed to be getting very thin, and on examination the physical signs of phthisis were discovered. She now had attacks of petit-mal very frequently and became very emaciated. She died on the 21st of December, 1875, having been unconscious the preceding day for 6 hours. The post-mortem examination was made 12 hours after death. The calvaria was removed, and the convex surface of the brain examined in situ. On stripping off the dura mater, the left hemisphere was seen to be much wasted, and the arachnoid membrane over its surface was in places thickened and opaque. The wasting was chiefly noticeable in the frontal and parietal regions. The texture of the hemisphere was evidently altered, for, to the touch, the left was hard and firm, while the right was elastic and apparently normal. The left middle lobe was much reduced in size, measuring only 1 inch transversely, the right measuring 2¼ inches in the same direction. There did not appear to be excess of fluid in the left subarachnoid space, but as some drained away while removing the calvaria, the defect may be thus accounted for. The brain was then removed and found to weigh 28¾ ounces. A quantity of fluid drained away during the process of extraction. A depression as large as a small pear could now be seen on the convex surface of the posterior half of the left hemisphere. On slicing through the brain transversely, the left lateral ventricle was found to be enormously dilated, measuring 4¼ inches in length and 2 inches in breadth at its posterior (most dilated) part, the right ventricle measuring only 3¾ inches, and 1 inch in the corresponding directions. The depression above mentioned was now seen to be due to the roof of the left ventricle falling inwards. Its upper wall in this situation was not more than ¼ inch in thickness, the roof of the right measuring ¾ inch. In consequence of the dilatation of the left ventricle, the left choroid plexus in its course outwards dipped downwards one inch anterior in position to the right. Comparing the two hemispheres, it was found that the right measured 7 inches in length, and 2½ inches in breadth, but the left only 6¼ inches longitudinally and 2¼ inches transversely. The right hemisphere weighed 15¼ ounces, the left only 5¾ ounces. The convolutions on the right side were exceedingly coarse, while those on the left side were smaller than usual for a child of her age. The white matter of the anterior and middle lobes on this side was reduced to a mere line. Unfortunately I did not examine the corpus striatum and optic thalamus of the two sides.

Examining now the cranium, the left side was found to be much thicker than the right, and the internal surface presented marked differences on the two sides. Thus the left anterior fossa was more prominent than the right, the left anterior lobe of the brain being correspondingly depressed inferiorly. The left middle fossa was much smaller than that on the opposite side and was pear-shaped, while the right was more quadrangular. On measuring the two, the left was found to be 1½ inch in length and 1¼ inch in breadth, the right measuring 2 inches and 1¾ inch in the same directions. The left posterior fossa was shallow. The left cerebella fossa was larger than the right. The lungs presented cavities in the apices, and patches of tubercle and caseous material were scattered through the substance of both.

The cerebellum was not specially examined, but from the fact that the left cerebellar fossa was larger than the right, there is no doubt that the left lobe of the cerebellum was the larger of the two.

The third case, A. H., æt. 16 years, was admitted into the Clapton Asylum, May 5th, 1875, and died November 12th, 1875. Her father had died at the age of 33, of typhoid fever; the mother was alive and healthy. There was no family history of neuroses, but the aunt on the mother's side had died of phthisis. Both parents had been temperate, and were not connected by consanguinity. There were three other children who were physically weak, but who were unaffected mentally. The patient is said to have been mentally sound when born, and nothing was specially noticed (except some spots on the body, when the child was a month old, due to the father having had syphilis before her birth) until four years of age when the child had a heavy fall, and the right side of her body became paralysed. Her intellect, however, was not noticeably affected until the age of 12 years, when she became subject to fits (cause unknown). These have continued ever since, but are slight in character. Since the onset of the fits she has become dull. On admission she was found to be emaciated and the right side of the body was paralysed. She could speak indistinctly, was quiet in disposition, but dirty in her habits. Her mental capacity was small. She could only read words of two letters, spell A B C, add to 6, count to 40, and recognise two colours. She was unable to dress, or take her meals without assistance. She went to school daily, but made little progress. She was very feeble, and frequently had attacks of diarrhœa and tonic contractions of the right side, no doubt epileptic in character. Later on, twitchings of the lip were noticed, and the left side of the body sometimes became affected. She died exhausted, November 12th, 1875. At the autopsy, the body was found emaciated and the right arm flexed on the forearm. The cranium was much thicker than normal. (I find I have not noticed whether it was thicker on one side than the other.) The brain weighed 29½ ounces, and was smaller than normal for a child of her age. The convolutions were flattened, and the sulci diminished in depth. There was a large excess of subarachnoid fluid, especially over the left hemisphere of the brain. The arachnoid membrane was opaque in places, especially over the site of the subjacent vessels. On slicing through the brain, the lateral ventricles and the fourth were seen to be much dilated with fluid, though some had drained away, the left lateral ventricle being apparently the fuller of the two, so that the septum lucidum was a little to the right of the middle line. On comparing the two sides of the brain, the left hemisphere appeared smaller than the right, especially anteriorly. The corpora striata, optic thalami and cerebellum appeared normal and equal in size, but this may have been due to the atrophy of the hemisphere being small in amount.

From the cases just given, as well as those related by Van der Kolk, Dr. Taylor in Guy's Hospital Reports, and others, it appears that the usual appearances found post mortem are: thickness of the cranium, opacity and thickness of the membranes, effusion of serum into the subarachnoid space, sometimes into the ventricles and atrophy of one hemisphere, including the corpus striatum, optic thalamus and pons of the affected side. Since the fibres of the superior peduncles of the cerebellum undergo a complete decussation beneath the upper pair of the corpora quadrigemina, and those of the middle peduncles decussate in the pons varolii, while the fibres of the pyramids of the medulla have their well-known crossed direction, there is atrophy of the cerebellum and of the spinal cord on the opposite side.

The course of events appears to be this; first, there is, as the result of chronic inflammation of the meninges, or of the cortical substance, wasting of one side of the brain. To compensate for this the skull becomes thickened, and serum is poured out beneath the arachnoid and into the ventricles. Then, since those parts of the brain which are connected with motion are wasted, the limbs whose action is governed by them are imperfectly nourished and become atrophied.

With reference to the microscopical appearances found in atrophy of the brain, a case is quoted by Dr. Major in the Journal of Mental Science' for 1879, in which especial attention was paid to this point, with the following result.

The cortex of the convolutions of the affected side was seen to be reduced in thickness, and under a low power seemed to be made up of small round cells of uniform size, here and there only a pyramidal corpuscle being observable, in marked contrast with the appearances in the cortex of the opposite hemisphere. Examination with higher powers showed that the nerve-cell elements were extremely few, as well as small, ill-developed, and deficient in branches. The intercellular matrix of the neuroglia was denser and coarser than in the right hemisphere and under normal conditions. In the internal white matter the nerve fibres were nearly absent in some places, the whole tissue being represented by numerous Deiter's connective tissue cells with their dense intercommunicating net-work, naked nuclei and occasional vessels. The atrophied lobe of the cerebellum showed a condition of thickness and wasting of the outermost grey layer, few and ill-developed cells of Purkinje, and excess of connective tissue corpuscles.

The spinal cord was not examined by Dr. Major, but was so by Dr. Taylor in a case related by him. There was shrinking of the anterior grey cornu and of the antero-lateral column of the affected side, but the number and size of the ganglion cells of the anterior cornua were almost identical. The cells on both sides were identical in colour after staining, in structure, outline, and processes, with those of the opposite healthy side. There was nowhere any degeneration of the nerve fibres in the white columns, and the dorsal and lumbar regions were distinguishable in no respect from those of a healthy cord.

ON THE ALTERATIONS OF CORTICAL EXCITABILITY BY COLD APPLIED TO THE SURFACE OF THE BRAIN.

BY H. DE VARIGNY, M.D. (PARIS.)

ALTHOUGH several physiologists have already investigated this question, there does not seem to be any general understanding as to the interpretation of the results obtained. I have recently made some ten or twelve experiments concerning this *quæstionem vexatam,* and I made them—purposely—before acquainting myself with the conclusions reached by others, but knowing the methods on which they proceeded in their experiments.

M. Marcacci, an Italian physiologist, has investigated the subject, if not thoroughly, at least more so than any one else. His experiments are described, by himself, in the 'Archives Italiennes de Biologie.'[1] They were intended to be used as facts in support of, or—as the case might be—against, the theory of experimental excitability of the gray matter of the brain. The nervous tissue, and especially the gray matter, must be considered as the most exalted of all the tissues, the last to be evolved, the one the functions of which are the most perfect and complete. It must also be the most delicate, that is, the one injury to which is of most consequence to the organism, as well as the one most easily injured. It is known in a general manner that all tissues die rapidly when submitted to the influence of a certain degree of cold, or of heat: M. Marcacci believes that the same is true of the gray matter; and we all agree in this, although not being certain which degree of cold—for instance—is enough to deprive the gray matter of its vitality.

But we do not know positively whether cold applied to the surface of the brain in the way in which M. Marcacci applies it does really kill the brain-elements: in fact, I do not believe it does; nor do I believe I killed them in my own experiments conducted in the same manner as M. Marcacci did. This author says—and it is true—that by pulverising ether, or other liquids, such as chloride of methyle, on the brain surface, one may obtain 20, 30, even 40 degrees below zero (Centigrade).

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[1] Vol. i., p. 261, 1882. "Etudes Critiques et Expérimentales sur les Centres moteurs corticaux." See abstract in 'BRAIN,' 1883, p. 142.

It is true that if such liquids are pulverised upon a thermometer, the cold may be intense enough to bring the mercury down to –30° or –40°, but are we sure that the same process can freeze the brain surface? I have often pulverised ether on this surface during ten and twelve minutes continuously; I never found the gray matter frozen; the blood did not cease to flow in the vessels, and the temperature always came back to its normal degree in a few minutes.

The surface was certainly cold after pulverisation, but it never remained so. I think it must be a very difficult process to really *freeze* the cerebral matter, even to half or quarter of an inch in depth, on the living animal. After my own experiments I feel quite sure that I never froze the brain-surface, nor even was near doing so, and I do not think the case to have been different with M. Marcacci. He shows that the pulverisation of ether, etc., on the brain-surface does not interfere with the excitability of the same; it even seems in some cases that excitability is increased. M. Marcacci concludes that when the brain-surface is excited (with electricity), the motor results are to be ascribed not to the excitation of gray matter, since its movements are obtained even where this matter is [thought to be] frozen, but to the excitability of the subjacent white tracts. I have already discussed the results obtained by M. Marcacci, in my work on the electrical excitability of the brain:[1] I shall not go over them again here. But I must remark that this observer's conclusions do not logically follow from his experiments. He shows that motor results are still obtained when the gray matter has been submitted to the influence of cold: we admit this; it shows that if the gray matter is paralysed, the white one is not; but does it prove anything more? Does it show that the gray matter is necessarily and always unexcitable?

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[1] 'Recherches expérimentales sur l'excitabilité électrique des Circonvolutions cérébrales, et sur la période d'excitation latente du cerveau.' Paris, Alcan, 1884.

In my own experiments, I have worked on dogs, and used the ether-spray. The way I proceeded was the following. The dog being narcotised with chloral (intra-venous injections), and the brain being exposed, I ascertained exactly what strength of current was necessary to obtain a motor-reaction of the fore-leg, for instance, and also the time required for the occurrence of this reaction—that is, the time elapsing between the moment when the brain was excited and the moment the motor result began: in short, the latent time of reaction. When these two points had been accurately ascertained, I began pulverising ether on the brain-surface, doing all I could to refrigerate the motor-centre of one leg only, for instance, without injuring that of the other. When refrigeration had been going on during five, ten, or fifteen minutes, I ascertained whether the motor-centre could be excited, with what current strength, and with what latent time.

I shall quote here some of my experiments.

Experiment 1.—Spaniel dog; weight, about six kilogr. Young, not very lively. The motor-centre of the fore leg is easily excited by Dubois-Reymond's secondary coil at 5° Cent. The latent time is 0·07 second.

I pulverise ether during eight minutes. I then try to excite the motor-centre with the same current (5° Cent.), no result. I then push the secondary coil gradually from 5° to 0°. Unfortunately the experiment had to stop here: I did not ascertain how much time was required for the recovery of the excitability of the motor-centre.

Experiment 2.—In this experiment I obtained the same results as in the preceding; that is, the motor-centre lost its excitability after a few minutes, and could no more be excited by the current which was previously sufficient, nor by the strongest current that could be obtained with the coil. However, after a little while, some reaction could be obtained.

Experiment 3.—In this experiment I obtained a very interesting result. After having ascertained the two data necessary in this sort of experiments, I sprayed ether during eight minutes. During five minutes after this, the motor-centre was entirely unexcitable. But within a short time it seemed to react again, and about a quarter of an hour after the spray the current required was the same as before. As soon as I perceived that excitability had returned, I ascertained the length of the latent time, and to my great astonishment found it shorter than I had ever done before the pulverisation. Instead of 0·09 second and 0·08 second, I found 0·07 second and 0·06 second. It seems as if the temporary paralysis of the nervous elements had been followed by a kind of increase of energy or hyperæsthesia.

I need not relate any more experiments here, but may confine myself to giving the general results reached by me. Generally speaking, after a five to ten minutes' pulverisation, excitability disappears or only diminishes. It can always be made to disappear if the spray be kept on long enough. After some while, if the cold has not been too intense, it reappears slowly, and may become greater than it was before. At any rate I have never seen a five to ten minutes' pulverisation injure the *cortex cerebri* to such an extent that recovery of excitability—transitory perhaps—became impossible. M. Marcacci either must have used very strong currents, capable of exciting the white tracts when the gray matter was paralysed, or—as I should feel inclined to believe—did not really paralyse the gray matter. I do not see how to explain otherwise the interesting results this physiologist has obtained. I cannot say anything definite as to the length of the latent time after the return of excitability; I have generally found it about the same, sometimes a little longer, and once much shorter.

Clinical Cases.

CASE OF OCCIPITAL ENCEPHALOCELE IN WHICH A CORRECT DIAGNOSIS WAS OBTAINED BY MEANS OF THE INDUCED CURRENT.

BY VICTOR HORSLEY, B.S., M.B. (LOND.), F.R.C.S.

Assistant Professor of Pathology in University College, London; and Surgical Registrar to University College Hospital.

MY excuse for publishing a case of Encephalocele, a condition the nature of which is well understood, lies in the fact that in this particular instance, as in all hitherto published, the *details* of diagnosis were very obscure, and I therefore ventured to clear up the difficulty by stimulating the hernial portion of brain with the interrupted induced current. Wishing also to draw attention to the causes of failure in those cases where operation was attempted, it seemed best to take this opportunity of discussing the various methods and their results.

I am greatly indebted to the kindness of Mr. Marcus Beck for the opportunity of treating this case.

CLINICAL REPORT, SEPT. 22ND, 1884.

ERNEST JOHN O., No. 1921.—Admitted into University College Hospital on 21st September, 1883. Age six weeks.

Past History.—The child was born at full time, the labour was easy, and nothing abnormal noticed, excepting the tumour which was at the time of birth equal in size to half the child's head. Child was "jaundiced" three days.

Family History.—Patient was the fourth child in five years, the last child being 16 months old. The second child died, cause not stated; the remainder were healthy. The father (an ostler) was a well-developed, healthy young man. The mother was of average height, but thin and poorly developed. Patient's maternal grandmother and three aunts died of "consumption." Maternal uncle now in Brompton Hospital with phthisis Father's family healthy.

Present State, on admission.—Patient was a pale thin child, with a pendulous tumour attached to the occiput. All its life-processes appeared perfectly normal, except that its temperature (rectum) varied from 97° F. to 99·4° F. as a "normal" range.

Head.—The skull was very prognathous and scaphocephalic. the masto-temporal ridge and lambdoid suture very strongly marked, while the fontanelles were perfectly normal. The scalp was covered with soft hair, and hanging from the occipital region was a sacculate tumour, which covered the lower two-thirds of the occipital bone, and occupied about half the breadth of the bone in the middle line. The tumour was covered by skin, the surface of which was harsh in patches, and reddened all over, with a few enlarged veins showing, close to the attached border. It was also hairy (like the rest of the scalp) for about one-fourth of its surface, extending from the attached border. Moreover, on the right side of the sac there was a large firm cicatrix (congenital) which was 3 in. long by ½ in. broad, situated opposite the middle, and directed backwards and slightly upwards. The circumference of the sac at its greatest diameter was about 12½ in. The wall of the sac was composed of skin, and a strong, evidently fibrous capsule with trabeculæ crossing the cavity at different points. The occipital bone could be easily felt to be perforated by a hole apparently 1 inch in diameter, and occupying one-third of the occipital crest. Emerging from this hole, and projecting into the fluid which filled the tumour, could be felt a rounded mass, apparently 1¼ inch broad, but (owing to the fluid around it) its outline could not well be made out. The rest of the surface of the body, the skeleton, the muscles, and viscera, appeared perfectly normal.

Central Nervous System.

Cerebrum Cortex.—Child appeared to possess the average mental capabilities of its age. It slept soundly, but when awake there was ptosis on both sides, so that the eyes were only half open. It took little notice of things around, and rarely made any purposive movements. When the chest was tickled, it (basal ganglia?) made semi-coordinated movements (defensive) of all the limbs. When awoke from sleep, its limbs performed irregular movements of flexion and extension, and sometimes the whole trunk also.

There was constant rigidity, with contraction of the flexor muscles of the left forearm, and slightly of the arm also. The elbow was kept flexed at a right angle.

Medulla Oblongata. Respiratory Centre.—Respirations normal. Firm pressure on the sac caused arrest of respiratory movements in full inspiration.

Cardiac Centre.—Heart beat perfectly regular. Only slight quickening of the pulse could be made out on pressure of the sac.

Vaso-motor Centre.—Skin over trunk and limbs moist and warm. Tache cérébrale well marked over the whole body.

Vomiting Centre.—Patient never vomited.

Deglutition Centre.—Swallowing perfect.

Cranial Nerves.

1st. Patient apparently noticed odours such as peppermint, &c. (Substances employed were not so strong as to irritate the 5th nerve.)

2nd. Patient took no notice of bright objects held or moved about in front of the eyes, and the pupil was not noticed to vary under those circumstances.

3rd. The pupils were equal and very small, being from 1 to 1·5 mm. in diameter. It was very doubtful, moreover, if they reacted to light at all, or very sluggishly.

4th and 6th. Normal.

5th. Normal. Tactile sensibility and reaction to ammonia normal.

7th. Normal. Saliva secreted freely. Pressure on the sac caused also a copious lachrymal secretion.

8th, 9th, 10th, 11th, and 12th, appeared normal.

Spinal Cord.—Motion and sensation transmitted normally. All the visceral centres and reflexes were perfectly normal.

Convulsions.—It was found by Mr. R. J. Godlee, to whom the patient was first brought as an out-patient, that pressure on the solid mass into the interior of the sac caused a general convulsion. After its admission the following facts were observed:

Pressure on the right side of the peduncular process (passing through the hole in the occipital bone) gave rise to powerful contractions of all the muscles of the trunk and limbs, but most especially on the left side. In each group of muscles the sequence of events was the same, namely, clonic spasms passing into tonic contraction, this stage being often incomplete, with the existence of tremors as a result, the rate of the tremors being about that of ankle-clonus. The order of march of the spasms was face, upper limbs, trunk, and then the lower limbs.

Contraction of the upper group of facial muscles on both sides was first noticed, and this was followed by powerful contractions of the left lower facial muscles, the angle of the mouth on that side being drawn strongly upwards and to the left, while the nasolabial fold was very much exaggerated;

the right lower facial muscles also were seen to be contracting, but could not antagonise the left side. This is seen very well in instantaneous photographs (taken very kindly for me by Mr. Hyde Marriott) of the child while convulsed.

There were no movements of the eyes nor of the pupils following the contraction of the face; the upper limbs became flexed in all segments; the rigidity of the left, noted above, being exaggerated, and both limbs showing the tremor very markedly. As before said, most movement was on the left side (*i.e.* opposite to that pressed upon).

After the spasms had become fully developed in the upper limbs, they invaded the trunk muscles and caused flexion of the trunk, *i.e.* slight emprosthotonus, the movements being accompanied by powerful tonic contraction of all the muscles in the lower limbs, the result being that all the joints were fixed in a position of 90°.

Directly the pressure on the tumour mass was removed, the spasms left off, the limbs returning to rest after a few irregular movements. Pressure applied to the opposite side (the left) produced the same movements, but much less marked.

Simple pressure to the convex surface of the tumour caused feeble movements of the same kind.

Oct. 5.—In the afternoon of this day the sac was aspirated and 370 cc. drawn off. The chemical reactions, &c., will be given later, as they were almost identical with those shown by the fluid obtained from the tumour by subsequent aspirations. During the aspiration the child seemed absolutely unaffected in every way.

The intrasaccular portion of the brain could now be felt to be a bilobed mass with a rounded surface.

After the aspiration the tumour very slowly filled again, and by the end of seven weeks had recovered the size previous to aspiration, viz. circumference 16 inches.

It was believed that, as has often been recorded, a small process of the choroid plexus projected into the interior of the sac (this however was soon found to be erroneous), and therefore it occurred to me to try the effect of twisting the sac on its pedicle. This was easily done by marking quadrant lines on the sac, and by continuing them on to the head, a definite means of recording the amount of rotation was obtained. It was found possible to turn the tumour 45° without causing more than transitory uneasiness.

The rotation, however, had none of the desired effects of checking the refilling of the sac, which simply became more tense, and in three days the whole of the skin covering it was deeply congested and of a bright red colour.

The sac was therefore aspirated again, and 15 ounces with drawn; but three days later the circumference was 14½ inches, and on the next day the child vomited. The sac was consequently turned back, when all these symptoms disappeared. On December 1st the sac was again aspirated, and 26 ounces drawn off. When the sac was completely emptied, the cerebral mass was felt to be distinctly fluctuating on the right side. A hypodermic needle, with syringe attached, was passed through the wall of the large sac into this smaller one, and 2·4 cc. of a slightly blood-stained fluid was drawn off. (This fluid was of a pale straw colour, was faintly alkaline, inodorous, and had a faint saltish taste. It gave no precipitate on boiling, but Millon's reagent threw down a cloudy precipitate, which turned brick-red on heating.)

During the following 24 hours, about 2 ounces of fluid collected in the sac. Twelve days later (December 12th), the sac was aspirated for the fourth time, and 17 ounces withdrawn.

The advisability of endeavouring to check the formation of fluid (see remarks) rendered it very necessary to know if possible what portion of the encephalon was protruded through the hole in the occipital bone. From a case[1] published at the time, it appeared very possible that the mass consisted of the corpora quadrigemina, and it occurred to me at once that this point could be cleared up, by applying the interrupted induced current to the tumour.

To the primary coil of an ordinary Dubois-Reymond coil with interrupter, a pint bichromate cell was attached, and small ball-pointed (¼ inch diameter) electrodes were fixed to the binding-screws of the secondary coil. The strength of current employed could just be borne by the fingers, the secondary coil being 4 cm. distant from the zero point (zero being the strongest). The following striking results were obtained.

(1.) When the current was directed *horizontally* through the mass, *no result* was obtained.

(2.) When one electrode was placed on the upper surface and the other on the lower surface, and the current consequently sent *vertically* through the hernia cerebri, there followed *rapid conjugate deviation* of both eyes towards the side stimulated. The conjugate deviation to the right, however, was not so marked as the movement to the left, which was very sudden. If the electrodes were taken off, the eyes returned slowly by jerks to the middle line again. If the electrodes were kept in apposition, the deviation of the eyes was not maintained for more than 10 seconds. Beyond salivation, the current caused no other symptoms, no change in the pupil nor in the rigidity of the left arm. Patient showed no sense of discomfort from the current.

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[1] Sutton, 'Pathological Transactions,' 1883, p. 18.

It was consequently assumed that the portion of brain involved was the corpora quadrigemina.

On December 20th the sac was aspirated, and 23 ounces of fluid drawn off; and again on December 28th, when 18 ounces were taken away.

In the hope of causing the sac to shrink, the posterior half was constricted off with a tape ligature, and into this portion was injected (December 28th)—with 18 minims of iodine solution—Pot. iod. gr. xx; iodine gr. x; glycerine ℥j.

The injection had no apparent effect on the child.

On December 30th, however, it was found that the wall of the sac was sloughing opposite the point of constriction, and therefore the much-delayed operation of cutting away the larger portion of the sac was commenced.

Although the patient was perfectly conscious, he did not exhibit any sense of discomfort when the harelip-pins used to temporarily close the sloughed opening were passed through the skin; consequently the following operation was performed without previously anæsthetising the child. As a precautionary measure, an india-rubber band was passed round the pedicle of the tumour to control hæmorrhage, but this was taken off almost directly.

A free vertical incision was made over the middle of the sac, the interior of which was found to be filled with a blood-stained serous fluid. In the posterior half of the sac the iodine had excited a little inflammatory disturbance, producing flakes of fibrine and soft trabeculæ, passing across the cavity, while the wall of the sac itself was œdematous and the surface hyperæmic.

The posterior half of the sac was then cut away freely with the knife, so as to leave two oval lateral flaps. It was now seen that the cavity was lined by the dura mater, in short, was part of the subdural space; for closing the aperture in the bone was a second much smaller sac, almost round, formed by a bluish membrane, and enclosing the hernial portion of the encephalon (see Fig.). It was clear that no good could result from opening this second sac, so an attempt was made to stop the "secreting" power of the dura mater lining the large sac, by sponging its surface over with chloride of zinc solution (40 grs. to ℥j). The line of incision was then closed with about thirty interrupted fine silk sutures; the wound was dressed with carbolic gauze and iodoform wool, the whole operation being performed under the spray. At the end of the operation, which lasted about 40 minutes, the child was very collapsed, unconscious, and cyanotic: 4 hours later, at 7 P.M., patient could not swallow, so an enema of milk and brandy (ʒj) was administered. This revived the child so far, that by 3 A.M. it was warm (temperature 101°), and taking its food readily.

On the next day, January 1st, 1884, the sac was found to be half full of fluid, wound looking perfectly quiet; the next day the sac was found to be distended, so a small incision was made into the left side, and slightly turbid serum allowed to escape, the opening being kept patent with a slip of protective. During this and the next day the patient gradually got more drowsy, and refusing food, it died on January 4th. After the operation the temperature rose gradually in 12 hours to 101·8, it then varied from 99 to 103·2 being 103 one hour before death, and 103·4 a few minutes after.

Note on the General State of the Patient.—From the time of admission to its death it took its food very readily; but in spite of that, it steadily emaciated, the loss of fat (of which tissue there was but little on admission) being followed by wasting of the muscles. During the two months preceding its death the emaciation proceeded very rapidly, and for the last month the food passed per anum but slightly altered by digestion. Alteration in its diet made no obvious effect.

Analysis of the Fluid drawn off from the Dural Sac.

The general appearance of the fluid and its reactions were so constant, that one description will suffice for the various quantities.

The fluid was proved to be albuminous, and it did not contain sugar nor extractives.

Fluid was of a pale-straw colour, clear, watery, inodorous, and had a saltish taste. The reaction was weakly alkaline (fixed). There was no deposit, and sp. gr. = 1006—1015.

(1) Boiling produced a very faint cloud, insoluble in acetic acid.
(2) Addition of H_2O, nil.
(3) Addition of alcohol threw down a white precipitate partly redissolved in excess of H_2O.
(4) Addition of ·2 per cent. HCl, nil.
(6) Potassic ferrocyanide and acetic acid gave a dense, curdy precipitate, insoluble on boiling.
(7) Millon's reagent gave a pink precipitate, turning crimson red on heating.
(8) Silver nitrate gave a curdy white precipitate, soluble in ammonia.
(9) Boiling with Fehling's solution gave only a dirty brown precipitate.

(10) There were no extractives nor urea.
(11) Estimation of phosphates by uranic nitrate:

Date.	Quantity.	Percentage of phosphoric acid.
October 10th	370cc.	·011
November 25th	320cc.	·013
December 1st	728cc.	·017
" 11th	476cc.	·02
" 20th	644cc.	·028
" 28th	504cc.	·028

NOTE.—It will be seen from this table of analyses, for which I am indebted to my friends, Mr. Hyde Marriott and Mr. MacCabe, that the proportional amount of phosphoric acid lost on each occasion gradually increased in amount as the case progressed, in fact, up to December 11th the increment was almost exactly ·003 per cent. This interesting fact should no doubt be considered in relation to the gradual emaciation of the patient; and the figures are here given in full, in hope that future observers may throw some light on the question.

Post-mortem, 26 hours after Death.—Rigor mortis almost passed off. Body extremely emaciated and very anæmic. The fingers of the left hand were still rigidly flexed on the palms, the thumb lying outside the forefinger. The sutured wound had healed perfectly by first intention, except just below the middle where a few stitches had been removed to improve the drainage. On laying the sac open it was found to contain a small quantity of sero-pus, while the sac wall itself was œdematous, being about ¼ inch thick, and its inner surface (dural) was acutely inflamed, and scattered over it were flakes of fibrinous exudation. The large sac was adherent to the inner arachnoidal one over the proximal third of its surface.

The arachnoidal sac projecting into the bottom of the dural cavity was tense, and while the upper part was of a yellowish colour over about one-fourth of the whole circumference, the remainder was dusky with a bluish-black tinge (post-mortem decomposition).

Skull.—The soft parts were now stripped off the skull, which was found to be apparently normal, exhibiting no true synostosis, the only unusual point about it being the size of the fontanelles, which were smaller than usual, but still preserved the ordinary appearances. The margins of the fontanelles, sutures, &c., appeared thick and pale, but afforded no evidence of congenital syphilis.

In the middle of the occipital crest there was an oval hole, through which the hernia had passed. This hole measured 1 inch long and ½ inch broad, covering almost one-third of the middle of the occipital ridge. The margin of the hole was thin, being bevelled off on both surfaces, but its edge was smooth and round. Rest of skeleton perfectly normal.

Meninges.—The dura mater was normal but thin, especially the tentorium. It was adherent by firm fibrous tissue to the margin of the hole in the occipital bone, and although thin, passed out to form the innermost layer of the sac wall, the tissue intervening between it and the skin being œdematous. The arachnoid also was quite normal, except at the margin of the hole, to which it was firmly united, and also to the dura, although the inner surface of this extra-cranial arachnoidal sac was covered with a layer of fibrinous exudation.

Sinuses.—Normal, except posteriorly, where the longitudinal divided at the apex of the lambdoid suture into two branches which ran down on either side of the occipital hole above described, and terminated in the lateral sinuses in the usual way, but opposite the middle of the cerebellum.

Central Nervous System.

The brain was exposed by cutting away the side of the skull and sac, as indicated in the figure, which is a photographic reproduction of a sepia drawing made life-size by careful measurements, and then reduced in the camera to two-thirds.

Cerebrum.—The cerebral cortex on the outer surface presented the normal fissures, but the secondary sulci were very numerous and complicated. Only the occipital lobe presented any peculiarity, and that lay in the fact that posteriorly the tips of both lobes were merged into the dilated corpora quadrigemina, the hernia cerebri. To make this clearer, it will be better to state at once, that on opening the arachnoidal sac of the encephalocele (before opening the skull) there appeared in view two ovoid masses, fixed by a narrow pedicle, which passed through the hole in the occipital bone to the encephalon. Of these two lobes, the superior, marked (N) in the figure, was a thin-walled sac formed of soft nerve-tissue, with a smooth fibrous exterior, and containing a turbid fluid with particles of degenerated tissue floating in it. This sacculated mass was found to be fixed to the pedicle by very firm connective tissue, continuous in front with the arachnoid and pia mater, which filled the sulci of the hemispheres, and helped to roof over the lateral ventricles, while posteriorly it wrapped round the pedicle of the rest of the encephalic mass (T), covering it, and continuous by its under surface with the sheet of tissue partly roofing the 4th ventricle. On vertical section this lobular mass (N) was found to exhibit, especially towards its fixed end, soft pink-grey tissue, somewhat resembling nerve-tissue. On microscopical examination, however, this was found to be exceedingly embryonic, consisting of neuroglia, with a few scattered small round corpuscles lying in distinct cell-spaces, forming a cortex, and beneath this was a layer of scarcely developed fibres.

The fibrous tissue forming the rest of the wall of this lobe was extremely vascular, evidently largely composed of pia mater, the vessels forming a rich plexus, mainly venous.

Possibly this mass represented the enormously enlarged pineal body, as it was only continuous with the rest of the cerebrum by the fibrous sheet, arachnoid, and pia mater above described; moreover, it fitted into the angle between the cerebral hemispheres and the corpora quadrigemina, and finally there was no other pineal body to be found. The inferior lobe of the hernia cerebri (T) proved to be the roof of the aqueduct of Sylvius bent backwards, so that the corpora quadrigemina were drawn out into the saccular form shown in the figure, the upper surface of this lower lobe being directly continuous with the posterior and inner aspects of the tips of the occipital lobes of the cerebrum. (In the figure this connection, together with the occipital and temporo-sphenoidal lobes of the left hemisphere, are cut away, so as to expose the Pulvinar. Pv.)

The sulci on the median aspect of the hemisphere were normal, except over the occipital lobe. Here, behind the parieto-occipital fissure, the sulci were arranged at right angles to the line of that fissure, and, running downwards and backwards, were lost among the fissures which marked the surface of the hernial lobe (T); shallow indications of these could be seen at the free extremity of the mass (see Fig.).

The aqueduct of Sylvius which formed the cavity of the lobe (T) was very narrow and flattened. On vertical section, the whole thickness of this lobe was clearly nerve-tissue in direct connection with the cortex of the occipital lobe of the brain, and on microscopical examination this lobe forming the corpora quadrigemina was found to consist wholly of nerve-tissue forming a cortex, with, underneath it, a layer of white fibres running forwards to the floor of the 3rd ventricle. This layer of course formed the wall of the central canal, and was covered on the inner surface by ependyma.

The Cortex (of the lobe T) presented characters intermediate between the structure of the cortex of the supra-marginal convolution (*vide* infra) and the slight remains of nerve-tissue found in lobe (N). Thus the basis consisted of a well-formed neuroglia, which in the medullary portion showed numerous round nuclei. In this ground-substance were scattered very numerous round corpuscles in cell-spaces. Immediately beneath the outer surface of the lobe these became fewer in number, but there was no corpuscle-free layer as in the cerebral cortex (*vide* infra). The vessels in this part ran at right angles to the surface dipping in from the pia mater, and were accompanied by very narrow perivascular. lymph spaces. All the vessels were engorged with blood.

The medullary portion was made up of horizontally directed fibres with parallel blood-vessels.

Microscopical Examination of the Cortex of the Left Supra-marginal Gyrus.—The sections of this gyrus showed a perfectly normal (adult) condition of the fibres of the corona radiata, and also of the outermost layer of the cortex (*i.e.* submeningeal), so that further description of these parts would be superfluous. Of the intermediate part of the cortex (which was of the usual depth) there was the normal neuroglia, in which were scattered both angular and round nerve corpuscles, all varying in size from four to twelve times the diameter of a red blood disc. They were arranged in a definite layer immediately beneath the outermost submeningeal layer, but beyond a slightly marked similar arrangement just above the fibres of the corona there was no further indication of layers. The vascular supply and direction of the fibres was quite normal.

The Basal Ganglia presented normal appearances on section.

Commissures.—The corpus callosum was represented only by a thin layer of white nerve tissue roofing the lateral ventricles (*vide* also supra). None of the transverse commissures could be made out. The fornix was normally developed.

The Cerebellum consisted of lateral lobes, normally developed and united in front by middle peduncles (white) of fair size, but posteriorly the vermiform processes were solely represented by two lateral rounded masses which projected into the cavity of the 4th ventricle, and were only separated from one another by a thin layer of pia mater.

The 4th Ventricle.—Normally bounded in front, and at the sides, was covered posteriorly by a strong sheet of fibrous pia mater and arachnoid.

Spinal Cord.—The only abnormal appearance in the spinal cord was the persistent embryonic shape of the central canal. This in the cervical enlargement still presented the well-known isosceles triangular outline, with the basal ends of the sides bent so as to be almost perpendicular to the base. In the lumbar region the canal was an irregular trapezoid, directed obliquely across the antero-posterior axis of the cord. Throughout it was lined with the normal epithelium. In some sections from the lumbar region was noted a condition I have also seen in the adult, namely, a small process of neuroglia usually opposite the posterior median fissure (pushing into the central canal for about ·5—1 mm. in depth. This process is not covered by the epithelium of the central canal.

With the exception of the alimentary tract, the viscera were normal, and there were no further developmental abnormalities.

Stomach.—Contained clotted milk, and abundant gastric secretion.

Intestines.—Both small and large were extremely pale, soft and thin. One large intestine contained clotted milk that was scarcely altered by digestion, only stained with bile.

Liver was pale, margins of lobules yellowish.

General Summary of the Post-mortem.—The death of the patient was clearly one from inanition due to the gradual failure of the alimentary tract to assimilate nutriment. The local inflammation in the sac of the tumour caused no symptoms beyond rise of temperature, and did not lead to general encephalitis. The outer sac had already adhered over the proximal third of the inner one, and the closed portion of the wound had healed by the first intention, so that local repair had proceeded very rapidly.

Remarks.

Beyond the minor point of interest, already referred to, in the composition of the fluid drawn from the sac, the questions of diagnosis and treatment alone call for attention.

Diagnosis.—The first case of occipital encephalocele which chiefly attracted notice to the subject was an observation of Lallement's,[1] on a patient aged 23 years, a young woman. In this instance, which was a small version of the one described above, the examination simply led to the discovery of a fluctuating tumour, and an exploratory incision was made into the sac, with the result of exposing an arachnoidal sac containing two lobes of the cerebellum. Although this was not opened, the patient succumbed in eight days to an attack of acute encephalitis and meningitis, no doubt of septic origin, judging from the brief but graphic history. This case illustrates very well the real difficulty of diagnosis in this condition, viz. (1) the presence or absence of a portion of the encephalon in the sac; and (2) the nature of the same. Obviously the former of these divisions of the question must be first determined, and supposing ordinary physical examination, such as palpation, translucency, &c., to fail, aspiration should be resorted to at once, the sac completely emptied, and the existence of a hernia cerebri determined. There should be no fear of awkward symptoms following aspiration. In the beginning of this century, Callisen[1] speaks of this trivial operation as quite safe, and in the case above reported, the only effect on the patient (even when the sac was absolutely emptied) was a very beneficial one. The lethargy which characterised it before usually disappeared at once, and the child crowed, moved its limbs and fed readily. (As a practical point it may be noted that the wall of the sac being inelastic, the fluid tends to ooze through the aspiration puncture, even when covered with collodion. It will be found that a fine harelip-pin and a loop of carbolised silk meets the occasion, by stopping the flow and healing the aperture.) With regard now to the position of the brain herniated, it is clear that in event of failure of ossification, in some portion of the occipital bone we may have protruded either the cerebellum,[2] the corpora quadrigemina,[3] or the occipital lobes,[4] all three together, on any combination of two, this being the commonest condition.

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[1] Richeraud, 'Nosographie Chirurgicale,' t. 2, pp. 396–322; 1815.

Under these circumstances the position of the tumour will offer some guide to finding the nature of the hernia by observing the relation of the tumour to the tentorum cerebelli. When beneath this structure, the hernia is practically certainly cerebellar;[5] when opposite to it, as in this case, the protruded mass may be the corpora quadrigemina; and when above, then the posterior extremities of the occipital lobes.[6]

It is often, however, not easy to make out the exact position of the pedicle of the mass involved in the sac, and so deductions have been drawn from motor symptoms, either chronic or excited by interfering with the tumour. Thus, for instance, in several cases,[7] as in the present one, there has been noted tonic contraction of the flexor muscles of one or the other upper limbs. In my case it was the left which first passed into this state, and very much later near death the right became similarly affected. In Lawrence's case just quoted, the sides were reversed, but the march of the rigidity was very much the same. This unilateral (in commencement, at any rate) condition may perhaps be due to irritation of the crura from the tension produced by the dragging backwards of the tumour. That this occurs was beautifully seen on the inner aspect of the cerebral hemispheres in the present case, where, as described above, the sulci are directed along the lines of the tension. This harmonises too with what is known concerning the mode of origin of this condition (*vide* infra).

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[1] 'Chirurgicæ Hodiernæ,' vol. ii. pp. 512–514; 1800.

[2] Richerand, loc. cit.

[3] Sutton, 'Pathological Transactions,' 1883, p. 18.

[4] Breschet, Obs. xxii., 'Archives générales de Médecine,' 1831, t. 25 and 26, p. 452 and p. 74.

[5] Breschet, op. cit., Obs. xiii., xxvi.

[6] Ibid. Obs. xxii., xxii., xxiv.

[7] Ibid, Obs. xiii.; also Lawrence, 'Med. Chir. Transact.' 1857, p. 307.

A more equivocal symptom is the convulsive stage which can be elicited by pressure on the whole tumour, or better (*vide ante*) on the side of the hernia cerebri. In a case reported by Wedemeier,[1] pressure caused loss of consciousness as well as convulsions; while Bruns[2] describes in addition, turning of the eyes, deep respirations, weakening of the pulse, sopor, twitchings, vomiting and choking; all these symptoms disappearing immediately the pressure is removed. From the detailed description given before of the similar phenomena as observed in the present case, and from the quotations just given, it is obvious that very little aid can be expected from such symptoms to help in localising the portion of brain protruded. This opinion is also supported by an observation by Job à Meeckren[3], who described a case in which the patient was two years old, and in which pressure on the sac caused syncope and loss of sight. It was found post-mortem that the dura mater (*sic*) closed the foramen in the skull.

The very general nature of the symptoms points to their finding origin in merely sudden increase of encephalic pressure, and in the present case this was proved to be so by squeezing only the distal portions of the sac, when the same phenomena were elicited, but at the same time not to so marked an extent.

Appreciating fully then the uselessness of mechanical stimulation, there remains electrical, and provided that in any particular case the efferent fibres from the hernial portion of the encephalon are intact, and the said portion well developed, we have, I believe, in the induced current an adequate means of diagnosing the part involved.

We might expect that stimulation of the corpora quadrigemina would always elicit movements of the eyes, while stimulation of the median portion of the cerebellum would produce in addition to movements of the eyes extension of the neck, &c., and irritation of the occipital lobes probably nil. In the present case only the eyes moved to the same side as that stimulated, and nothing else occurred. This was noted by Ferrier[4] as a result of irritating the pyramid of the median lobe of the cerebellum in monkeys, but in addition there ensued movements of the head and neck. That stimulation of the corpora quadrigemina produces movements of the head as well as the eyes, is of course well known, but that movement is in the same direction as that taken by the eyeballs, and not at right angles as in the case of the cerebellum. However, of course, but little stress can as yet be laid upon this point, which is only brought forward to suggest an additional means of diagnosis.

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[1] Wedemeier, 'Journal für Chirurgie u. Augenheilkunde,' 1826, Bd. 9, p. 152.

[2] Bruns, 'Die Chirurgischen Krankheiten in Verletzungen des Gehirns,' &c., 1854, pp. 695–720.

[3] Quoted by Ferrand, 'Mémoires de l'Acad. de Chir.' t. 5, p. 863; T. 13, p. 102.

[4] 'Functions of the Brain,' 1876, p. 98.

Treatment.—The treatment adopted in this case was that indicated by Callisen (loc. cit.), with the additional attempt to diminish the secretion of fluid by twisting the vessels of the pedicle.

It is perfectly clear that aspiration per se is of no use, and aspiration followed by pressure is equally futile. To arrive at some definite course of rational treatment, we must for a moment consider the probable mechanism of the affection. Seeing that we find every stage between meningocele and encephalocele, from the slight bulging of a fontanelle to the most complicated palatine hydrencephalocele,[1] it is no wonder that Adams[2] in 1883 suggested that the first stage consisted in a meningeal protrusion, the pressure in which was kept up by increased secretion within, and that the cerebellum followed, half pushed, half dragged out. The theory of adhesion in utero to the amnion at a very early period, evidence of which is found by those supporting it, in the scars observed on the cutaneous surface of the sac (*vide antea*), accounts readily for the primary production of the cranial defect, and the more so, since, as in the present case, there is not always premature synostosis of the sutures. Of course when the latter malformation occurred[3] it would form a strong predisposing cause. Doubtless in different cases, each one of these causes or combinations of them may be in operation.

Treatment clearly then should be, first to stop, if possible, the meningeal hernia, and the first successful case[4] was treated by pressure applied by means of a leaden plate. The tumour in this instance was very small, and easily dealt with, but in more severe cases, as in the above, it would obviously be quite incompetent. Since the sac formed by dura mater and arachnoid continues to secrete serous fluid and yet the lymphatics running towards the base of the skull are blocked in the pedicle of the tumour (by simple pressure at first, at any rate), it is obvious that the natural direction (as I regard it) of the circulation of the cerebrospinal fluid is interrupted, and therefore must collect in the closed sac, producing all the symptoms resulting from pressure on the encephalon. The only course open to relieve this state of things would appear to be this: cut away the posterior half of the sac, remove the dura matral lining of the peripheral portion by rapid dissection (the application of strong antiseptics caused, I think, too much inflammation of the sac in my case), and bringing the edges together in the middle line by a continuous suture of chromic catgut. Drainage must of course be provided for, by a free opening at the lowest point. By the employment of continuous suture, I am confident the operation could be performed in fifteen minutes, and shock thus greatly avoided. Of course it must be carried out with the strictest antiseptic precautions, as the mortality of septic meningitis makes perfectly clear the folly of opening the dura mater except under those conditions.

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[1] Virchow, 'Die Krankhaften Geschwulste,' 1863, p. 188 et seq.

[2] Adams, 'Dublin Journal,' 1833.

[3] Breschet, loc. cit., obs. xv.

[4] Salleneuve, quoted by Ferrand, q. v.

The sac when healed would have united firmly over the arachnoidal sac covering the encephalocele (as it was rapidly doing in the present case even when the patient could scarcely be said to be assimilating any nutriment). When healed in this way, no further secretion would occur, pressure would prevent further hernia cerebri, and adhesions fix everything in situ.

It is perhaps not chimerical to suggest, that a bony covering could be obtained for the tumour by transplantation of periosteum, &c.

The fact that of 44 cases collected by Lawrence the cerebrum was normal only in 5, and further, that all the recorded cases I have met with where the patients attained to an age of discretion were imbeciles, renders the successful application of treatment doubtless somewhat dispiriting; still, that of course should not weigh in discussing the merits or demerits of any particular line to be adopted.

NOTES ON A CASE OF PURE APOPLECTIC BULBAR PARALYSIS.

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APOPLECTIC bulbar paralysis, when associated with hemiplegia, is not of unfrequent occurrence; but when the bulbar symptoms alone exist, the case, from its rarity, is of exceptional interest. The reason is obvious: the anatomical relationship of many of the motor nuclei in the medulla is so close, that an extremely localised lesion is required in order to limit the mischief to two or three motor nerves. Apoplectic bulbar paralysis is usually associated with alternate hemiplegia, or with paralysis of the upper, lower, or of all four extremities. The disease, as a rule, is initiated by an apoplectic seizure, varying from a momentary loss of consciousness to a profound attack, which in some cases is instantaneously fatal. The region attacked is perilous, and very little deviation of the lethal force is sufficient to convert a circumscribed lesion, capable only of producing a local paralysis of a limited extent, into an occasion of immediate death. In looking over the literature of the subject I have been able to find only three cases comparable with the one about to be described, as regards the limited area attacked, and the uncomplicated nature of the onset of the disease.

In 'Guy's Hospital Reports for 1870,' Dr. Wilks mentions a case in which the patient, whilst at dinner, fell off her chair without losing consciousness, and at the same moment lost the power of speech. She could not swallow without great difficulty, and had constant dribbling of saliva from the mouth; the mental faculties and powers of locomotion were undisturbed. The patient lived for two or three years after the attack, but never spoke again. Hérard, in the 'Union Médicale,' 1868, reports the case of a man aged 68 who was healthy up to the time of the attack. He awoke one night and found himself unable to speak; both tongue and lips were paralysed. The left corner of the mouth was lower than the right; food fell out of the mouth, and saliva constantly ran over the lower lip. The act of swallowing was not greatly interfered with, but the movements of the vocal cords were imperfectly performed. The intelligence was unaffected, and the limbs retained their normal power. The patient gradually improved, and was able to resume his work, although nine months after the attack a trace of the injury still remained.

A third case by Charcot is reported by Huret in his 'Tribut à l'histoire de l'Embolie des artères vertébrales, 1873.' It is that of a woman aged 68, who, three years before the attack in question, had an attack of left hemiplegia from which she had quite recovered. One day, without any apoplectic seizure, she lost her speech; she understood all questions quite well, but could not answer them. The right corner of the mouth hung down and could not be moved; the movements of the tongue were partially lost. The patient had considerable difficulty in swallowing, the food returning partly through the nose. There was no paralysis of the extremities. The right pupil was smaller than the left; the heart-sounds were normal. The symptoms gradually improved, and in three months the patient could almost be said to have recovered; the right corner of the mouth, however, remained somewhat lower than the left. In addition to the above cases, Duret quotes one of Voisin's in which bulbar paralysis occurred suddenly, but I have not been able to meet with a detailed account of the symptoms. The following case came under my own observation.

M. D., a married woman, 42 years of age, rather above the average build, of pallid complexion, active in habits, and free from any syphilitic taint, eleven years ago had an attack of rheumatic fever. Since the fever, she has, on two occasions at least, suffered from general dropsy of cardiac origin; at the time of the attack about to be described, she was in her ordinary health. She had not previously suffered from any cerebral symptoms. The patient attributed the attack to a state of excitement (induced by some domestic trouble), into which she fell shortly before the seizure.

On the 27th of October, 1883, whilst walking in the street with another woman, the patient suddenly became conscious of having lost the power of speech. This occurred without any apoplectic seizure whatever; she did not fall, nor had she any pain or dizziness in the head, nor did she even momentarily lose consciousness. Within an hour after the occurrence of the symptoms, the patient walked without difficulty to the Salford Royal Hospital, a distance of about half-a-mile, and there saw Dr. Haslam, who has since informed me that her mental faculties were then quite clear, and that her bodily movements, except those of the tongue and mouth, were perfect. She went home, and remained there for a few days, when, on account of the difficulty experienced in administering nourishment, she was admitted into the hospital, and came under my care. On November 1st, 1883, her condition was as follows:—Mental faculties quite clear; she responded intelligently to questions either by signs or by writing on the slate. The tongue, somewhat coated, lay flaccid on the floor of the mouth, and could not be moved in the slightest degree, by any effort of the patient. The soft palate was flabby, and hung loosely down, bringing the pillars of the fauces somewhat forwards. The pterygoids were completely paralysed, no lateral movement of the lower jaw being possible. The masseters and buccinators did not appear to have suffered so much; the jaws could be brought together, but with less than the usual power. The muscles attached to the lower jaw and the hyoid bone were not affected, the elevation of the larynx in the act of deglutition being normal in amount. The orbicularis oris was completely paralysed; the patient was totally unable to purse the lips as in the act of whistling, nor could she kiss the back of her hand, nor blow her nose; the latter disability, of course, was partly due to the condition of the tongue and soft palate, as well as to that of the orbicularis oris. The levator anguli oris, levator labii superioris, and zygomatici of the right side were completely paralysed; the corresponding muscles on the left side were not fully paralysed, but their motility was very slight; the patient was only able to impart the least flicker of movement to the left corner of the mouth, and none whatever to the right. The corners of the mouth were equal in height. The ocular and upper facial muscles were altogether unaffected; the patient was able to close the eyelids, and to resist any attempt to open them by force. The trapezius, sterno-mastoid, and all the other muscles of the trunk and extremities were altogether unaffected. The nerves of special sensation were severally tested, and found to be in their normal condition. There was no singing in the ears, nor was there any sensory paralysis. All the facial muscles responded normally to both constant and interrupted currents; the reflex of the soft palate was not materially, if at all, lessened. The only sound the patient could utter was a nasal "a," being the primitive sound produced by the vocal cords without glossal or labial modification. A laryngoscopic examination revealed nothing abnormal about the action of the vocal cords. Saliva ran continuously out of the mouth, but there did not appear to be any excess in amount; the overflow was simply the result of inability to swallow, and to close the lips. On being asked to swallow, the patient took some water into the mouth, and after a momentary pause, accompanied by a peculiar, expectant look, she slowly turned back her head so as to allow the water to flow towards the fauces. The result was an explosive cough, which forcibly ejected the fluid both from mouth and nostrils. Occasionally the attempt was more successful, and the fluid wholly or in part reached the œsophagus; the act of swallowing, however, was always followed by more or less cough. It was observed that in the early stage of the disease the patient could swallow aërated fluids with greater chance of success than still fluids. The temperature was normal, the pulse 72; there was no increase in arterial tension. A slight mitral systolic murmur was heard over a limited area. The urine contained neither albumen nor sugar.

For the first day or two after admission the patient was nourished by nutrient enemata, afterwards she was fed with the œsophageal tube. On the eighth day after admission the tongue had recovered a slight amount of motility, and as the patient strongly objected to the use of the tube, she was allowed to try to swallow small quantities of finely divided meat. When she concentrated her attention on the act of swallowing, and performed it in a deliberate manner, as a rule she was successful; but if she attempted to swallow quickly, the food was promptly ejected from the mouth by an explosive cough. Fourteen days after the attack she could protrude the tongue about a quarter of an inch beyond the teeth, could slightly purse the lips, and swallow prepared food with tolerable certainty; she was, however, quite unable to masticate. She has continued to improve, and her present condition, six months after the commencement of the attack, is as follows:—The tongue can be protruded about half an inch beyond the teeth, but she cannot lift it to the roof of the mouth, nor can she curl it up at the sides. She can purse the lips pretty well, but without much power; she cannot smack them, nor can she blow her nose. She can swallow soft pulpy food better than fluids, but cannot masticate even bread. There is now a trace of lateral movement in the lower jaw, and the muscles on the left side of the face have considerably improved; the corresponding muscles on the right side have also recovered a limited amount of motility. The patient can make some attempt at articulation, and occasionally one can make out the meaning of what she endeavours to say, but there is no real speech. She complained a few week ago of giddiness when walking, but has improved in this respect. She also complains of weakness of vision. In an early stage of the case I examined the discs without finding anything abnormal, at that period the patient made no complaint about her vision. Later on, my friend Dr. Glasoott kindly examined them and found the retinal veins engorged, especially those of the right side, the margins of the discs were ill-defined, but there was no retinitis. The deep reflexes of the arms are now slightly more marked than in the normal state; in the early stage nothing abnormal in this respect was observed. The compressive power of the hands tested by the dynamometer equals 25 kilogrammes for the right, and 23 for the left; the difference is too slight for unilateral paresis, and is more probably due to the usual predominance in muscular power of the right hand over the left. The excess of reflex just mentioned would probably be caused by slight inflammatory processes set up in the immediate neighbourhood of the focus of injury; the disturbance of the retinal circulation might proceed from the same cause.

There is not much difficulty in localising a lesion such as this, the symptoms at once point to the posterior part of the rhomboid sinus—to a region embracing the nuclei of the hypoglossal, facial, and motor branch of the fifth nerves. The facial nucleus proper is removed from that of the hypoglossal; but the inferior facial nucleus is about on a level with, and closely external to it. It is supposed that the accessory facial nucleus exclusively controls those branches of the facial nerve which supply the lower facial muscles: it is therefore probable that, as regards the facial nuclei, the accessory nucleus only was attacked, the supply to the upper facial muscles on this account not being interfered with.

As regards the nature of the lesion, acute myelitis may be omitted, as this disease, although it may occur very suddenly, requires at least several hours to develop, and usually runs a rapidly fatal course. The present case, on the contrary, was instantaneous in onset, and the tendency from the first was to improvement. There is then left the choice between blood-extravasation, thrombosis, and embolism. Against extravasation is the early improvement, and the improbability of the occurrence of such a very limited effusion. Whatever was the cause of the lesion, it was of a peculiarly circumscribed and selective character; it attacked a series of nuclei in the same order that they are attacked in progressive bulbar paralysis, the course of which follows a certain definite sequence. It is difficult to imagine that extravasation—the amount and localisation of which would be determined by contingency—could produce such a methodic arrangement of symptoms. It would rather be expected that other motor centres would be attacked, and that an anomalous chain of symptoms would result; still the rupture of a miliary aneurism might produce a sufficiently localised extravasation. Thrombosis presupposes, more or less, the existence of preliminary symptoms: the gradual closure of a vessel would reduce the blood-supply of the region nourished by the vessel, and would probably produce prodromata as dizziness or paræsthesiæ, both of which were absent in this case. Here again the elimination cannot be absolute, for thrombosis does at times develop symptoms suddenly and without previous indications. Embolism seems to be the most likely causation: the attack of rheumatic fever and the subsequent heart mischief give a certain colour to this supposition. It is probable that an excited action of the heart, induced by the domestic trouble before mentioned, led to detachment of a minute vegetation from the left side of the heart, which was carried with the blood stream along the vertebral and basilar arteries and finally lodged in one of the small branches. Almost all the arteries of the medulla are terminal arteries, and therefore such a clot would give rise to one or more of those well-known wedged-shaped infarcts, with their basis directed towards the floor of the fourth ventricle, and including in their sphere of action the nuclei before mentioned.

There is one point worthy of note—the two groups of muscles most profoundly attacked in this case, the same muscles which are first attacked in progressive bulbar paralysis, viz. the extrinsic lingual muscles and the orbicularis oris, were the first to show signs of returning motility, and have relatively undergone the most marked improvement. The nuclei concerned are the accessory facial and the hypoglossal: the hypoglossal nucleus receives its blood-supply from terminal branches of the anterior root-artery and of the anterior lateral artery. Both the hypoglossal and the inferior facial nuclei are supplied by branches from the artery of the median raphé. Assuming embolism to have been the cause of the paralysis, it is possible that the main blood-supply of the nuclei affected was cut off by the embolus, producing for a time total cessation of function, which was afterwards restored within certain limits by collateral supply.

CASES OF CHOREA.

BY W. B. HADDEN, M.D.

A. *Complete loss of Articulation in Chorea.*

Case I.—A. L. æt. 7, female, was admitted into St. Thomas's Hospital, under Dr. Bristowe, on January 10th, 1879. She had never had acute rheumatism. Eight months before admission she had been under treatment on the surgical side for chronic abscesses around the knee-joint. Her present illness began two weeks ago, with movements chiefly affecting the right side. On admission, the chorea was found to be general, but most marked in the face, tongue, and right side. There was great difficulty in articulation, and also some dysphagia. She had tinea tarsi on the right side, and several chronic indolent ulcers on right leg and thigh, apparently the result of bone disease. No cardiac murmur was detected. On January 19th it was found that she could not speak. Next day she made an attempt, said "yes" and "no," but refused her name. On January 22nd she had recovered speech, but the words were jerked out, and syllables often mispronounced and slurred. The day following she was very quiet, and speech was again entirely lost. The muscles of the face were less affected, and the tongue protruded more easily. The temperature the previous evening was 102°·2. On January 24th, that is two days later, she could say a good many words quite distinctly, protruded her tongue quietly, and was altogether better. On February 3rd she was again unable to articulate, and the movements of the legs were markedly choreic. On February 18th, she could say several words distinctly, and repeat her name. From that time she steadily improved, and was discharged cured on March 19th.

Case II.—S. G., æt. 14, female, was admitted under Dr. Stone, on May 27th, 1879. She had never had acute rheumatism, but there was a history of fright just before present attack. On admission, she had general, but not very violent, choreic movements. The speech was slow, but not otherwise affected. She could not feed herself, and swallowed with difficulty. On

June 26th she was found to have lost all power of articulation, and this continued for more than a week. I found that the right hand was decidedly weak, and that there was anæsthesia of left arm and slightly of left leg. There was a soft systolic murmur at the apex of heart. She was discharged relieved on August 6th.

CASE III.—S. R., æt. 12, female, was admitted under Dr. Harley, on May 30th, 1883. She had had acute rheumatism when six years old. In January, 1883, she had another attack which was very severe, and was followed by chorea and loss of speech. On admission, the movements were general, but most marked on right side. She could not articulate, but the next day she said a few words. On June 14th it was noted that the patient not only does not talk, but makes no attempt to frame words, and did not even try to speak to her mother. She protruded the tongue when asked. Since the previous day she had screamed much, and was very restless. On June 16th she could talk a little, and from that time continued to improve. There was a systolic murmur at the apex. She was discharged cured, on July 19th.

Remarks.—It is a curious and suggestive fact that, in all the cases the choreic movements were most marked on the right side, and in one that there was decided paresis of the right arm. The inability to articulate seems comparable to the paralysis of limbs, which may either precede or follow an attack of chorea. But whether the actual cause lay in the brain itself, or in the medulla, or in the exhausted muscle, I am not prepared to say.

B. *Anæsthesia in Chorea.*

CASE I.—E. M., æt. 20, female, was admitted under Dr. Harley on May 6th, 1879. She had had convulsions during dentition, and acute rheumatism ten years ago. Six weeks ago she had a severe fall, and was much frightened. Three days later, movements began in the left arm, and were limited to that part until ten days before admission, when the chorea became general. About this time she was said to have had another fright, and since then her memory has become impaired, and she has had delusions. On admission, there were general choreic movements, but the left side was much more affected than the right. The left arm and leg, too, were very weak. The speech was but little impaired, and there was no difficulty in deglutition. These was total anæsthesia of left arm and leg, and partial anæsthesia of right arm. Her mental condition was much impaired. She did not know how long she had been in the hospital, but she answered readily when spoken to, and had no delusions. There was no cardiac affection. On May 9th she became very restless and violent. She now had distinct delusions. She thought she was going to be poisoned, and that she would never see her parents again. At this time she was being treated with conium. On May 28th she developed well-marked symptoms of conium-poisoning, and accordingly the drug was omitted. The same treatment was subsequently resumed, but without benefit. On June 22nd she tried to drink a bottle of carbolic acid, but was fortunately observed. On July 9th, however, she was found with an empty bottle, labelled "poison," which had previously contained ʒ vij of a solution consisting of morph. acet. gr. $\frac{1}{4}$, and atropiæ sulph. gr. $\frac{1}{60}$ in ten minims. It was not until two hours and a half later that any symptoms were observed. It was then noticed that she tottered when walking. She soon went to sleep, and four hours after the solution was taken, she was apparently sleeping quietly. Half an hour later she was heard to groan feebly, the lips were blue, and the respiration seemed to have ceased. The extremities were very cold, the pupils dilated, the conjunctivæ insensible. Artificial respiration was kept up for twenty-six hours, and stimulants administered. No spontaneous effort at breathing was observed for twenty-four hours. She was comatose throughout, but could easily be aroused by shaking. Her mental condition was more unsatisfactory than before. She subsequently acknowledged to taking the poison, but said she poured some away. She was discharged on July 22nd, and I believe went to an asylum.

Case II.—E. R., æt. 15, female, was admitted under Dr. Ord on Jan. 22nd, 1879. Her mother and eldest sister were liable to faints, but there was no decided neurotic or other taint in the family. The patient had never had acute rheumatism or scarlet fever, but for the last six months she had been under treatment for enlarged glands in the neck. A month ago she began to have twitching of the left hand, then the face became affected, and lastly the leg. On admission, there were slight choreic movements of the head, left arm and both legs. There was also paresis of the left arm. It was found that there was anæsthesia of the left arm and leg. A systolic murmur was audible over the pulmonary artery. She was discharged cured on March 19th.

Case III.—E. B., æt. 16, female, was admitted under Dr. Bristowe on June 25th, 1879. She had had rheumatic fever, and five-and-a-half years ago was attacked with chorea. She had a second attack three months later, and from that time until May 1st, 1879, there were occasional tremblings of the hands, feet, and face. On that day she had a fresh attack of rheumatism, which was followed by chorea. The movements affected both hands and feet, and slightly the face. The power of the right hand was much impaired, although the movements were less evident here than on the opposite side. She did not feel when a needle was thrust into the right leg, but flinched when touched with a pin on the left leg. Her mental condition was fairly good, but occasionally she called out in her sleep. There was a mitral systolic murmur. On July 8th, sensation began to return in right leg, and on the 18th, there was only a little difference between the two sides. She was discharged cured on July 27th.

Remarks.—I am inclined to think that there is no rule as regards the distribution of the anæsthesia in chorea. In Cases I. and II., as well as in two or three others which I have observed, the impairment of sensation was on the side most affected by the movements. In Case III., however, it will be seen that the anæsthesia was chiefly in the right side, whereas the choreic movements were mainly left-sided.

C. *Hysteroid Convulsive Attacks in Chorea.*

C. W., æt. 15, male, was admitted under Dr. Bristowe on January 16th, 1882. On December 20th he suddenly became faint and sick, and fell on his face. He was helped up, but a few minutes later he fell again, and began to kick about. He had some half-dozen such attacks on the same day. The choreic movements are said to date from this time. The boy was found in the casualty room, lying on a couch, with the body rigid, and working his arms and legs about. He was quite conscious, but emotional, crying readily when asked about himself. His mother said that before the fit he complained of not being able to swallow, and of tightness in the throat. Later, he was found to have characteristically choreic movements, his speech was imperfect, there was no anæsthesia, and no heart affection. On January 22nd he complained of a lump in his throat, and very soon he jumped up, and would have fallen out of bed if he had not been caught. He rolled over on his face, sobbing. When he was turned over, his back became quite rigid, and he worked his limbs about. He did not lose consciousness. He was discharged cured on January 31st.

Remarks.—Emotional disturbance is common enough in chorea, but the hysteroid convulsive attacks which occurred in this boy are probably rare. Unfortunately there is no record of the family history, nor of the previous history of the condition of the patient.

A CASE OF DORSAL PACHYMENINGITIS.

BY HOWARD H. TOOTH, M.B.,

Assistant-Physician to the Metropolitan Free Hospital.

THE case about to be related, though somewhat deficient in clinical details, yet is possessed of some pathological interest.

The patient G.G., aged 60, a compositor, was admitted into one of the surgical wards of St. Bartholomew's Hospital, on October 14, 1883, suffering from simple fracture of the lower third of the left femur, owing to the accident of falling downstairs.

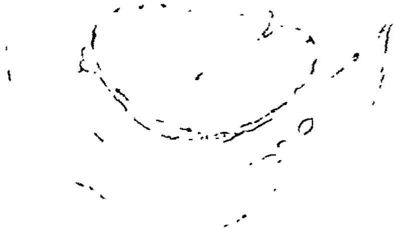
The notes of the case as regards spinal symptoms are unfortunately rather meagre. They may be summarised as follows.

For ten years at least he has suffered from progressive paresis of lower extremities, which increased to such a degree that for five years he has been unable to get about without assistance. During this period and till death, has been subject to excruciating shooting-pains in the lower limbs, most severe in the left. There were also frequent pains of the same character in the left side, about the position of the lower ribs. No atrophy of muscles. Great reflex excitability of both legs, with sudden painful spasms at night for some years. Bowels always very costive, but no loss of control over rectum. Has had to use a No. 10 catheter for over five years, unable to pass water without it. His wife says that he has been extremely irritable and capricious in temper lately. He is the father of five children, two healthy, three died at about fifteen months "of convulsions and teething."

No history of syphilis, direct or indirect, could be elicited.

Oct. 14*th*, 1883.—On admission, considerable rigidity of both legs was noted, most obvious in the sound limb. Thigh adducted. Intense reflex excitability; so much so in the fractured limb, that splints had to be discontinued and the limb laid on a pillow. Distinct loss of sensation in both limbs. Upper extremities quite natural.

Oct. 19*th*.—A large bedsore discovered over sacrum on right



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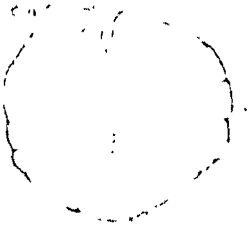

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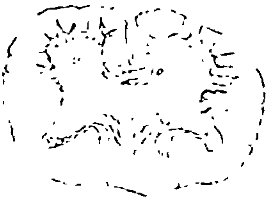

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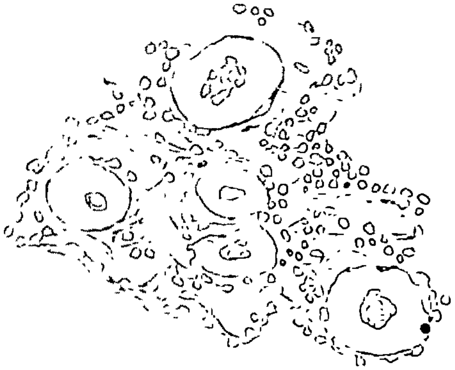

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side. Cystitis. Urine highly coloured, offensive; sp. gr. 1020. Alkaline. Albumen and pus.

Oct. 20*th*.—Pain in right shoulder, with some difficulty in raising the arm.

Oct. 23*rd*.—Shoulder better.

Oct. 25*th*.—Cystitis improved. Diarrhœa.

Nov. 14*th*.—Bedsores over both great trochanters.

Nov. 30*th*.—Abscess in scrotum. Opened.

Dec. 28*th*.—Going on well till this morning, when he had a rigor at 6 A.M. Temp. 103·4°.

Jan. 13*th*, 1884.—Another rigor at 6 A.M. Temp. 102·6°; unconscious muttering, delirium.

Jan. 31*st*.—Sore on back worse. Edges undermined, spines bare. Right great trochanter exposed.

Feb. 11*th*.—Died. The temperature during this period was always above normal at night, sometimes 103°–104°, often above normal in the morning.

At the autopsy, made on Feb. 12 by the surgical registrar, Mr. Bowlby, to whom I am indebted for the spinal cord, the cadaver was found to be well nourished. No atrophy of muscles. Brain quite natural. Thoracic organs natural. Liver, spleen, stomach and intestines quite natural. No signs of visceral syphilis. Kidneys enlarged, and suppurating in places. Ureters dilated, and bladder much thickened.

Spinal Cord, Macroscopic appearances.—Whole arachnoid-cavity much distended with clear fluid. At about the middle of the dorsal region, involving a length of about 7 cm., the membranes are fused together into a tough white leathery mass, about 5 mm. thick. This thickening involves rather more than one-third of the circumference of the sheath, and is adherent closely to the cord along the posterior half of its circumference. The cord at this point is much flattened out, measuring only 5·5 mm. antero-posteriorly. Here also the cord is twisted and distorted, being drawn round from right to left, so that the thickened mass, though adherent over the posterior segment, lies in the bony canal rather to the left side. It is not adherent to the bony canal, nor is there any disease of the vertebræ. The posterior roots are involved in the membranes on both sides. Anterior roots natural.

Microscopic examination.—Cord hardened in ammonium bichromate solution for three weeks. Stained in picrocarmine and hæmatoxylin.

Mid-dorsal region (Fig. 1). *Membranes*—Thickened dura mater composes about one-fifth of the whole mass. The component bundles of connective tissue are separated by large numbers of leucocytes. Vessels are numerous, the walls of which are not markedly thickened. Between the dura and pia mater is a mass of fibrous material making up quite ⅘ of the whole. This consists in its outer ¾ of bundles of large wavy structureless fibres, cut in all directions, but mostly running parallel to the fibres of the dura. There are numerous lacunæ containing nuclei, but no vessels. As the inner boundary is reached, the fibres become smaller and the lacunæ larger, containing numbers of leucocytes. Here numbers of blood-vessels make their appearance (Fig. 4). The vessels here, and to a somewhat less degree in the succeeding sections of the cord, have remarkably thickened hyaline walls. All distinction between middle and outer coats is lost. The elastic lamina is undistinguishable in most of the sections. In some the intima is considerably thickened. There is in some of the vessels a distinct proliferation of nuclei of the intima.

Cord.—Flattened antero-posteriorly. Right half smaller than left. Relation of parts distorted, as seen in figure.

In the white matter, the whole section shows softening with destruction of nerve fibres. The softening is much more marked on the anterior or unattached segment than on the posterior adherent segment. This must be due to latter being in a measure protected from the acute inflammatory processes spreading in from the spinal canal. The thickened vessels pass into the cord from the pia mater, and are scattered plentifully in cross-section over the whole section.

The right anterior horn of the grey matter is much attenuated; only one or two ganglion cells can be found, and they are highly degenerated. Left anterior horn also greatly affected, though less so than right. Some four or five ganglion cells are visible, but all showing more or less granular degeneration. Left posterior horn elongated and attenuated to a much greater degree than the right. The left posterior roots show in section proliferation of nuclei with considerable disorganisation of the nerve fibres. The right posterior roots look natural. Both show great hyaline thickening of the vessels, which in this part reach their highest degree of degeneration.

Upper dorsal region (Fig. 3).—Fibrinous thickening of pia mater. Marked softening of the white matter all around the periphery of the section, with complete disappearance of nerve tubules in the softened area, except on each side of the anterior fissure.

As the grey matter is approached, the nerve fibres make their appearance, and here the neuroglia becomes considerably thickened and condensed. This is most marked in the posterior columns and reaches its greatest degree in the postero-median columns, where there is considerable sclerosis. Even here there are, however, many axis-cylinders. The ganglion cells of the anterior horns are wanting in sharpness and distinctness.

Cervical region.—To the naked eye the carmine-stained sections show slight deepening of colour marking the postero-median columns, and under the microscope there is a marked increase of the neuroglia. Except for the peripheral softening, as in the preceding section, the cord is natural here.

Upper lumbar region (Fig. 2).—Shows a considerable increase of connective tissue in the posterior columns. This is most marked in the middle $\frac{1}{3}$ of the posterior radicular zones, where large branching fibres pass from the posterior horns towards the posterior median fissure. The white matter between this and the transverse commissure shows similar connective tissue-increase, but to a less degree. The same may be said of the lateral columns close to the grey matter.

Sections taken lower still in the cord show less and less departure from the normal.

Remarks.—In spite of the lack of syphilitic history, there can, I think, be no doubt of the specific nature of the affection in this case. It probably originated at a gumma of the dura mater in the subarachnoid space, which subsequently took on the present fibroid condition.

A noteworthy feature in the case is its chronicity. The fatal termination might have been indefinitely postponed, had it not been for the accident, whereupon an acute meningo-myelitis was lighted up with the accompanying cystitis and bedsores.

One cannot help speculating on the possible good results of an early and vigorous mercurial treatment in this case. Certainly the effects of such treatment are not encouraging in the later stages, as detailed in a somewhat similar case reported lately by Eisenlohr.[1]

Though the neuroglia changes in the posterior and postero-median columns above the lesion are probably of a secondary nature, the same cannot be said of the similar changes in the same regions below. We have here a distinct but early stage of sclerosis. The nerve fibres are diminished in quantity, but their structure is little altered.

At the same time one cannot overlook the well-marked changes in the small arterioles. These are shown in Fig. 4, which is taken from a part of the thickened meninges, and is a rather strongly marked type of the vascular changes in a greater part of the cord. The lumen is much diminished, and in some there is a distinct proliferation of the nuclei of the intima; beyond this, however, there is in the majority of the vessels no thickening of the intima to the great degree described by Heubner,[1] Drs. Greenfield, Gowers, Barlow and others.[2]

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[1] 'Neurolog. Centralblatt,' Feb. 15, 1884; and Abstracts in the present number of 'BRAIN.'

In the dura mater, or rather the most peripheral part of the area of pachymeningitis, we find the vessels not increased in thickness, but the perivascular spaces crowded with leucocytes. Such a condition has often been described;[3] and in an extreme degree by Dr. Buzzard in the last number of 'BRAIN.'

It is tempting to connect the two conditions, and to suppose the hyaline thickening to be a further development of the perivascular infiltration. If this position can be accepted, there is no great difficulty in connecting the fibroid changes in the neuroglia with those of the vessels, when the close communication between the lymph-spaces of the cord and those around the vessels is borne in mind.

Lastly, these vascular changes might be attributed by some to the condition of the kidneys; but I think the fact that they are most affected in the region of the syphilitic lesion is in favour of the theory of an extension of the so-called "gummatous processes" into the perivascular spaces.

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[1] 'Die luetische Erkrank. d. Hirnarterien,' 1874.
[2] 'Visceral Syph. Path. Trans.' V., xxviii.
[3] Moxon, 'Lancet,' Sept. 25, 1869. Bruberger, 'Virch. Arch.,' 1874, p. 285.

Critical Digest.

ON RECENT ADVANCES IN THE ANATOMY OF THE NERVOUS SYSTEM.

BY JAMES ANDERSON, M.D.

FROM the observations of Huschke it appears that the child brings with it into the world one third of the brain-volume it will ultimately possess. It developes the second third in the course of its first year of life, and the remaining third is gained before the twenty-first year. During this first year's rapid development the brain grows 400 ccm., that is more than one 1 ccm. daily, or a piece the size of a bean. Little was known of the histological details of this growth, till Exner showed that of medullated nerve-fibres the infant's convolutions contain practically none. Tuczek and Fuchs now confirm with some modifications the results obtained by Exner, and trace the development of medullated nerve fibres. The former[1] examined by Exner's process four brains; two of children born dead at full time, one of a fœtus of thirty-four weeks, and the other of a female child twenty-seven days old. The parts were placed ten hours after death in ¾ per cent. osmic acid solution, changed in two days, and examined on the sixth or seventh day. Dr. Tuczek summarises his results as follows:—

(1) In all the cerebral convolutions medullated nerve-fibres appear first in the medulla, then in the cortex; and the development of the same extends from centre to periphery.

(2) Medullated nerve-fibres appear earliest—sometimes before the end of the ninth month of fœtal life—in the medulla and cortex of the paracentral lobule, that is, in the median part of the anterior and posterior central (ascending frontal and parietal) convolutions. Next they appear in the occipital lobes and in parts of the Island of Reil. The rest of the cortex contains, in the infant, no medullated nerve-fibres. That is, the law of Flechsig, that medullated nerve-fibres appear first in the region of the pyramidal tracts and *corona radiata*, may be extended to the convolutions connected with them.

(3) The farther development of the medullated nerve-fibres progresses at an equal rate in the convolutions. In the case of the twenty-seven days' child, beyond the paracentral lobule, the central convolutions and the occipital lobe, no convolution contained

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[1] 'Neurol. Centralbl.' 1883, No. 20.

medullated nerve-fibres in its cortex. Even in the named convolutions these were contained only in the lower third of the grey cortex. Especially the tangential system of superficial fibres was completely absent.

(4) Medullated nerve-fibres are developed latest in the frontal lobe both on the vertex and the base. The straight convolution, the orbital part of the frontal lobe and the middle frontal convolutions contained in the twenty-seven days' child, even in the medulla, no medullated nerve-fibres.

Fuchs so far confirms the above results[1] that he finds no medullated nerve-fibres in either medulla or cortex of the infant's cerebral convolutions. In the medulla they appear first during the first month of extra-uterine life. In the superficial layer of the cortex they appear first during the fifth month; the second layer (Meynert) shows them first at the end of the first year; the third layer (fibræ arcuatæ) shows them in the seventh month; while the radiating bundles of the deeper layers show them in the second month. After the first year they increase steadily in number and calibre till the eighth year, when they are practically as in adults.

It has for some time been well known that the anatomical description of the spinal cord, as consisting of anterior, lateral and posterior columns of white matter with a double grey crescent in the centre, was a barren topographical division; convenient on certain occasions, as are the "artificial" classifications of plants, but useless as an instrument for scientific progress. We are now learning the same truth in regard to the brain. Dr. Hughlings-Jackson has in his Croomian lectures on the Evolution and Dissolution of the Nervous System, divided the nervous system not anatomically but physiologically into highest, middle and lowest centres, and he has spoken of the evolution of the cerebro-spinal functions from the most automatic to the least automatic, from the most simple to the most complex, from the most organised to the least organised. The anatomical results above detailed are in full agreement with his physiological and clinical generalisations. In such a division of the nervous system as Dr. Jackson has elaborated for its motor side, and in a similar division for the sensory side which, notwithstanding its greater difficulties, will doubtless in the near future be elaborated—we have the "natural" division of the nervous system as opposed to its "artificial" topographical division, and in the recognition of this division we have the promise of a steadier and more scientific advance, both in the physiology and in the pathology of the nervous system. Not that the new physiological subdivision of the nervous system will displace the old topographical. This it will no more do than will Dr. Jackson's classification of the diseases of the nervous system, as dissolutions to various depths of nervous system, displace the clinical classifications at present in vogue. The two divisions, topographical and physiological, will stand side by side, each useful in its own way, each giving useful help to the other.

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[1] 'Sitzungsbericht der Wien. Acad. d. Wissensch.,' July, 1883.

At a meeting of the Medico-Chirurgical Society on Jan. 22nd, a very distinct advance was made in our knowledge of the anatomy of the peripheral nervous system by a preliminary communication from Mr. Victor Horsley, in the course of which he demonstrated, by numerous and convincing specimens, the existence of true sensory nerves supplying nerve trunks—of nervi nervorum as opposed to the nervi vasorum, or vaso-motor nerves, already described as supplying the blood-vessels of the epineurium. These nervi nervorum come off at right angles from a primary bundle or cutaneous branch, and terminate, as Mr. Horsley's preparations showed, in end bulbs lying in the perineurium. They have, up to the present time, been concealed in osmic-acid preparations by the fat cells of the epineurium, but can be seen if, as Mr. Horsley suggested, the specimens be taken from an emaciated dropsical subject. This demonstration will afford a safe foundation of fact for any theories as to the cause and treatment of the lightning pains of tabes dorsalis or other neurotic affections.

The 'Brit. Med. Journal' May 24th, 1884, contains an abstract of a Lecture delivered by Professor Hamilton, of Aberdeen, in which he states some of the results of observations made by him, according to the method described in 'BRAIN,' July 1883. The lecture is necessarily given in an extremely fragmentary form, and we defer for the present any extended notice of the points contained in it. That it deals with sufficiently radical and important matter will at once be admitted, when we say that Professor Hamilton, from his observations, believes the corpus callosum to be not a commissure, but the decussation of cortical fibres on their way down to enter the internal and external capsules of the opposite side. The statement practically involves the reconstruction of central cerebral anatomy, and we expect with interest the publication of the lectures *in extenso*.

In the examination of the brain and spinal cord it is frequently difficult to say with certainty whether minute changes observable in the nervous tissue are due to post-mortem changes, to the effects of the hardening reagents, or on the other hand to commencing pathological alterations. The rule therefore with every careful observer is to note, in every case, the period that has elapsed between death and autopsy, the state of the brain and cord on removal, and the processes through which they subsequently pass. Having done this, he describes what he sees, without in doubtful cases deciding as to the exact nature of the minute changes observed. This is not altogether satisfactory, and hence we welcome as extremely valuable the results published by Dr. Richard Schulz[1] of the examination of the spinal cord in twenty subjects of various ages, who had shown no symptoms of spinal disease during life, and whose spinal cords were to the naked eye healthy. The causes of death and all the circumstances of the autopsy are carefully recorded. All the cords were alike hardened in Müller's fluid, frequently changed. They were then placed in gradually stronger and stronger, till finally in absolute alcohol.

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[1] 'Neurol. Centralbl.' 1883, Nos. 23, 24.

They were cut with Thomas's microtome in sections $\frac{1}{30}$ to $\frac{1}{40}$ mm. thick, stained with Borax-carmine, partly decolorised by acidulated (HCl) alcohol, and mounted in balsam.

Dr. Schulz discusses in detail the following appearances whose significance is more or less uncertain. (1) *Vacuolation of ganglion cells.*—This consists in the occurrence of two or three clear spaces in the protoplasm of the cells, the nucleus being sometimes visible, sometimes not. It occurred in two out of the twenty cases, and was accompanied by signs of slight imperfection in hardening. Dr. Schulz agrees with Charcot that this is an artificial appearance, and, from the difficulty of hardening the cord in cases of myelitis, he considers that it would be specially liable to occur in myelitis, as also in any condition where the ganglion cells are specially delicate. Leyden considers that vacuolation of the ganglion cells is pathological, and denotes myelitis. Drs. Pick and Rosenbach, who have criticised Dr. Schulz's opinion on this point in subsequent issues of the 'Centralblatt,' also believe that it is pathological, and the latter points out that it occurs in marked degree in the brain and cord of starved animals, and considers therefore that it comes under the head of a degenerative atrophy. (2) *Pigmentation of ganglion cells.*—This consists in a yellowish-brown pigment surrounding, and more or less concealing the nucleus. This was present in a high grade in four cases, aged respectively thirty-two, fifty-two, fifty-three, and seventy-six years; in a slight grade in eleven cases of middle age; while in five young subjects it was completely absent. Dr. Schulz therefore concludes with Charcot that this condition is a matter of age, but much depending on the nutrition of the individual; while it unquestionably occurs also as a pathological change in irritative processes commencing primarily in the neuroglia, also in progressive muscular atrophy and bulbar atrophy. By itself there is no reason to conclude that it is pathological, and Charcot considers that it is the concomitant of the tissue-degeneration and loss of muscular power which accompanies natural old age and the *senium præcox* of Virchow. (3) *Hypertrophy and sclerosis of ganglion cells.*—By hypertrophy of the ganglion cells is meant a swollen, cloudy, opalescent condition of the cell-substance with swollen processes. By sclerosis or atrophic sclerosis of the ganglion cells is meant a shrunken opaque or glancing appearance of the cell substance, with deficient processes. Dr. Schulz in almost all cases found the cells large, with sharp outlines, well-developed processes and distinct nuclei. In seven cases, however, varying from eighteen to seventy-six years of age, the outlines were not sharp, the cell-substance was hyaline in appearance and the nucleus was indistinct. In only two cases were the cells shrunken and hyaline in character. Hypertrophy or hyaline swelling of the cells is therefore not a matter of age, nor of post-mortem change; and while the higher grades are undoubtedly pathological (the result of irritative inflammation, as Charcot, Leyden, and Erb believe), seeing that these seven healthy spinal cords showed the change, it is unwise to pronounce on slight degrees of the change, more especially unaccompanied by other inflammatory changes. As to sclerosis of the ganglion cells, which usually accompanies only well-marked inflammatory changes, Dr. Schulz considers his cases too few to enable him to pronounce an opinion. (4) *Swelling and hypertrophy of the axis-cylinders.*—The normal diameter of an axis-cylinder, according to Charcot, is ·0033 mm., but in some cases it is as much as ·0099 mm. swollen and irregular in outline. This varicose condition has been observed in the retina, and by Virchow in yellow atrophy of the cortex, and in congenital interstitial encephalitis. All observers agree in regarding it as pathological, occurring in acute and chronic myelitis, in grey degeneration and sclerotic atrophy. Dr. Schulz found the axis-cylinders in eight cords not quite so sharply outlined at the periphery of the cord, this specially where there was slight swelling of the ganglion cells, but nowhere was there an actual enlargement of the axis-cylinder, and the change had quite the appearance of being artificial. Were the swelling of the axis-cylinder a change, artificial or *post-mortem*, Dr. Schulz considers that it would almost certainly have shown itself in some of his cases. He therefore inclines to believe that, when recognised, it may be considered a definite pathological change. (5) *Aggregation of round cells in and about the central canal.*—This Dr. Schulz considers in no way pathological. In some young spinal cords he found the central canal surrounded by a single layer of columnar epithelium; in others there was a growth of round cells around the canal, and in others the canal was completely blocked with round cells. (6) *Neuroglia.*—This in well-hardened specimens is delicate and sharply defined with distinct Deiter's cells. In some cases, however, Dr. Schulz found it not clear, and with no cells visible. This he considers artificial, as the condition was accompanied by swollen ganglion cells and ill-defined axis-cylinders. In six spinal cords, that is in 30 per cent., there were present thin laminæ of fibro-cartilage in the pia mater, and this not in old spinal cords only, but also in those of young adults. It is probably a concomitant of natural or of premature old age.

We have before us an admirable monograph by Dr. William Browning, of Brooklyn, on the veins of the brain and its envelopes. It consists chiefly of anatomical work done by Dr. Browning in Professor Braune's Laboratory in Leipzig, and will undoubtedly in future be the authoritative exposition of this subject. The work contains numerous additions to and corrections of our knowledge of the cerebral veins, one of the principal corrections being as to the course of the Vena Galeni. This it has been the custom to regard as passing directly backward into the straight sinus. A moment's consideration of the relative position of the tentorium cerebelli and the posterior part of the corpus callosum ought to have shown us that such could not possibly be the case, and this is clearly seen in the figure given by Dr. Browning, which shows that the Vena Galeni, passing backward and downward through the median part of the transverse fissure of Bichat, describes a circle upwards round the splenium or posterior part of the corpus callosum, and so at its termination in the straight sinus "the sinus rectus bears the same relation to the Vena Galeni that a tangent does to its circle." In other words, the Vena Galeni enters *against* the blood-current just as do the superior cerebral veins. Of this somewhat puzzling fact Dr. Browning gives a highly probable explanation in the backward development of the posterior part of the brain carrying its veins with it, and more especially by the late development of the corpus callosum extending backward, and so causing the Vena Galeni, as it were, to circumnavigate it. This explanation, as Dr. Browning states, is corroborated by the more straight course of these veins in the fœtus. Dr. Browning has also carefully examined the important subject of the anastomosis of the cerebral veins. As with the cerebral arteries, so with the cerebral veins, he has nowhere within the brain-substance succeeded in finding a venous anastomosis. For details as to the superficial anastomosis, we must refer to the original work, but we may state shortly that he proves an anastomosis, (1) between the dural and cerebral veins, (2) between the veins of the vertex and those of the base, and (3) between the external veins which join the *portio curvata* of the Vena Galeni and the veins of the base. This last fact shows that "Galen's system of veins is not isolated from the other cerebral veins," but this statement does not apply to the internal veins which run in the choroid plexus and velum. These anastomose in the velum, but nowhere beyond, and thus while obstruction of the straight sinus and of the short common trunk of the Vena magna Galeni can be compensated for, obstruction of both the straight sinus and the compensating basilar vessels, or of the trunks of the Venæ Galeni in the *portio recta*, before they are joined by these basilar veins in the *portio curvata*, means cerebral softening and death.

In the 'Neurol. Centralbl.' 1883, No. 10, Dr. Mendel describes a case of the Ape's Fissure (*Affenspalte*) occurring in the brain of an idiotic girl, eight years of age. Starting in the position of the parieto-occipital fissure just in front of the transverse occipital sulcus, a deep sulcus ran transversely out, ending behind the posterior extremity of the parallel fissure, and thus entirely separating the occipital from the parietal lobe on its convexity. This differs from the cercopithecus and cynocephalus in this region, only in the fact that in these apes the intraparietal fissure and the parallel fissure pass farther back, the first ending in the ape's fissure, neither of which was the case here. That it was really the ape's fissure Dr. Mendel believes was proved by the fact, that on the median surface it communicated with the calcarine fissure in the usual way. Dr. Mendel gives a list of cases where this peculiarity was present, some associated with mental disease, others not. He regards it as an arrest of development. The brain in this case was both smaller and lighter than normal. The frontal convolutions were very small, and the gyrus opercularis round the ascending limb of the fissure of Sylvius was very small, and (owing to absence of the ascending frontal convolution) was in direct continuity with the ascending parietal convolution. Regarding the question of the homology of the Affenspalte, whether it corresponds with the transverse occipital sulcus or with the parieto-occipital, or with both, as Schwalbe believes, Dr. Mendel has satisfied himself from vertical sections of the brain of cynocephalus sphinx that it corresponds with the parieto-occipital fissure, along with a fissure running forward from it in the depth, termed by Meynert the external occipital sulcus.

For teaching purposes it is frequently important to have the brain of natural size and in a state fit for handling. We make no apology therefore for quoting a process for preserving it given by Germa.[1] (1) The brain is hardened for fourteen days in a saturated solution of bichromate of ammonia. (2) It is washed and placed for one day in a mixture of carbolic acid 1 part, alcohol 1 part, water 8 parts, and glycerine 10 parts. (3) It is washed, and then allowed to dry in the air, and covered with three or four fine coats of varnish; the different parts being coloured, if necessary, with variously coloured metallic powders. (4) Over all is laid on a coat of painter's varnish, for the sake of preservation.

Notwithstanding that the function of speech has now for many years been a favourite subject of physiological and pathological observations, there still remains much to be done before we can be said in any complete sense to understand it. Dr. F. Raymond and M. Artaud[2] have recently made a painstaking and valuable contribution to the subject of the *Intracerebral Course of the Hypoglossal Nerve.* The groundwork of clinical fact and the inferences built upon it are so clearly and logically laid down, that it is difficult in abstract to do justice to their work, which involves a wider range than the actual title of their paper. In writing their paper, the authors had not been acquainted with that by Dr. Ross "On Glosso-labial Paralysis of Cerebral Origin," in 'BRAIN,' July 1882, and their results are therefore the more valuable as agreeing with his.

Affections of speech owing to paralysis of the muscles of articulation have been observed from (1) Cortical lesions; (2) Lesions of the Centrum ovale; (3) Lesions of the internal capsule and crus cerebri; and (4) Lesions of the Pons Varolii. That a lesion of the posterior third of the third left frontal convolution will cause aphasia is now a well-substantiated fact, but that a lesion of the inferior part of the ascending frontal convolution produces glossoplegia or paralysis of the muscles of articulation, is a fact not yet so well substantiated. Of this last the authors cite six cases. In four of these the lesion was located in the inferior part of the ascending frontal convolution, right or left, the result being convulsion or paralysis in the muscles supplied by the hypoglossal of the opposite side from the lesion; and the third frontal not being affected, in no case was the disorder accompanied by aphasia. In two cases both aphasia and glossoplegia was present, the lesions being bilateral and affecting both the inferior and ascending frontal convolutions.

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[1] 'L'Encéphale,' 1882, No. 4.

[2] 'Archives de Neurologie,' 1884, Nos. 20 and 21.

From experiment and clinical observation it would seem, according to Charcot and Pitres, that the inferior third of the ascending frontal convolution presides also over the movements of muscles supplied by the inferior branches of the facial, and recently M. Lépine has reported cases of permanent trismus accompanying lesions of this area, or of the subjacent white matter. It forms, therefore, the cortical origin of hypoglossal, inferior facial, and motor root of the trigeminus.

Tracing the hypoglossal fibres downwards, Dr. Raymond and M. Artaud cite seven cases, one recently observed by themselves, of glosso-labial paralysis resulting from injury to the inferior frontal fasciculus of Pitres, arising from the part of the cortex above designated. While the principal symptoms of the progressive bulbar paralysis of Duchenne, viz. paralysis of the tongue, lips and velum palati, were present in these cases, the differential diagnosis is comparatively easy. The symptoms simulating Duchenne's Paralysis are less marked, and differ in their mode of onset. In glosso-labial paralysis of cerebral origin the symptoms commence abruptly, frequently with an apoplectic seizure, accompanied by fall and loss of consciousness; and when the seizure passes off, the patient speaks with difficulty, and cannot move the tongue or approximate the lips. These symptoms are sometimes accompanied by facial paralysis, hemiplegia and intellectual defects, such as are observed in cerebral softening, or by convulsions and epileptiform attacks. In Duchenne's Paralysis the disease is progressive, attacking successively tongue, lips and soft palate, cerebral symptoms being absent; while on the other hand a group of bulbar symptoms are present which are wanting in glosso-labial paralysis of cerebral origin, viz. laryngeal (progressive weakness of voice) pulmonary (attacks of suffocation) and cardiac (syncope). The first disease is also distinguished by the presence of the reflexes, the absence of muscular atrophy, and the conservation of electrical excitability. Death results in the first from fresh cerebral hæmorrhage or softening, or from intercurrent disease of lung or kidney; in the second, from an attack of dyspnœa or syncope.

The fibres of the inferior frontal fasciculus above referred to as arising from the inferior part of the ascending frontal convolutions, pass downward in the internal capsule at the *genu* which divides its anterior from its posterior segment, forming what the authors term the "geniculate" fasciculus, bounded in front by the "intellectual" fasciculus and behind by the pyramidal" or motor fasciculus (from the limbs) and the "sensory" fasciculus. Without being able to render actual proof, Dr. Raymond and M. Artaud believe with a certain probability that the fasciculus of aphasia and that of glosso-labial paralysis pass down in different tracts through the internal capsule and the crus cerebri, the former occupying a position intermediate between the intellectual and geniculate fasciculi. As to the farther course downward of the cerebral fibres of the hypoglossal through the Pons, the authors quote three cases which lead them to believe that they occupy in the Pons the part internal and posterior to the motor pyramids.

Reviews and Notices of Books.

Les Maladies de la Volonté.—Par Th. Ribot. Deuxième Édition. Paris, 1884. 18mo., pp. 177.

The task undertaken by M. Ribot in this volume is to treat disorders of the Will from the same point of view and in the same manner that he has previously found so effectual in the case of disorders of the Memory. Dealing with a subject which is, as the author says, more difficult and less explored than the subject of the sister volume, the success that he attained in his former work is scarcely to be expected in this. Nor would such an expectation be realised. The book affords much pabulum for thought; it frequently compels our adhesion to its doctrines, and it maintains in us a steady conviction that the author is honestly facing his difficulties, which are neither few nor small. But it is by no means so clear, so convincing, nor so free from inconsistency as the author's previous work.

The most interesting and important part of the book is the Introduction, for in this chapter M. Ribot deals with the normal Will, and sets forth his own doctrine concerning both the nature of the state of consciousness that we term willing, and the nature of the physiological process which is the accompaniment, the physical aspect or substratum of the conscious state. This is of course the fundamental problem upon which the whole of the rest of the book depends; and when the method of its solution is known, the mode of treating the portion of the subject indicated by the title can be to a considerable extent foreeasted.

After mentioning the problem of free-will, merely to place it on one side as beyond and apart from the scope of his enquiry, M. Ribot postulates that dualism of which he is a steadfast champion, and states as the foundation of his doctrine the elementary principle that every state of consciousness has always a tendency to express itself—to issue in a movement, in an act. This principle is but a particular case of the general law that all nervous action is reflex. The most refined and original exercise of Will is but an immensely developed reflex action, accompanied by a peculiar state of consciousness. The continuous gradation that unites these extremes is divided by M. Ribot into three stages, the division being understood to be arbitrary. First, there are those mental states which issue in action with a certainty and rapidity almost equal to reflex action. Such are the passions and the "fixed ideas." Second, come those cases to which the term Will is usually applied—cases in which a resolution issues in an act after more or less deliberation; and the third group consists of abstract ideas, whose tendency to issue in action is very slight. Each more elaborate mental state is less apt to issue in action than the less elaborate states, because, in the first place, its anatomical substratum includes fewer and feebler motor elements; and in the second, because the substratum is, as a whole, less directly connected with the motor tract.

Will does not, however, manifest itself solely in movement. It is also displayed, and in its most striking and characteristic instances, in arrest of movement. That the voluntary arrest of movement is a higher development of the physiological process of inhibition, is sufficiently obvious, but as to the intimate nature of either process M. Ribot does not add much to our present knowledge. He takes, as is usual, the influence of the vagus on the heart as the simplest example of inhibition; but in the light of recent researches it becomes extremely doubtful whether this is so, and whether the inhibition of the heart's beat is not indeed an extremely complex and altogether exceptional instance of this process. The current notion of inhibition scarcely gives to it a position or a scope commensurate with its importance and its prevalence throughout the nervous system; although the doctrine of "loss of control," which implies the doctrine of universal inhibition, has long been domiciliated amongst us. If there is such a thing as excessive action of a nerve-centre, permitted by the removal of the function of the centre above, then it is only saying the same thing in another way to say that the superior centre normally inhibits the inferior centre. And just as we have already so modified our conception of co-ordination as to ascribe it, not as a special and separate faculty to a single centre or region of the nervous system, but as a universal attribute to every centre in every region, so we must extend our conception of inhibition. It is as absurd to speak of some centres as motor and others as inhibitory, as to speak of some centres as motor and others as co-ordinating. We must recognise that the power of inhibition is as integral and necessary a part of the function of every nerve-centre as the process of discharge or the attribute of co-ordinating. What is the meaning of the increase of reflex action in the lower section of a divided spinal cord, but that the inhibition of the nerve-mass above is removed from it? What is the meaning of the rigidity of a hemiplegic hand, but that the inhibition of the cerebral centres is removed which counteracted the nervous afflux that maintains the rigidity? From top to bottom of the hierarchy of the nervous system every centre inhibits those below, and is inhibited by those above. On any other supposition the hypothesis of over-action from loss of control is nonsense. The inhibition exercised by the highest of all centres upon the whole mass of strata below it, is the physiological side of the voluntary arrest of movement, just as the motor discharge of those highest centres is the physiological process in voluntary action. What the precise molecular mechanism may be by virtue of which the same centre can act on its subordinates, now as an inhibitory or restraining, and now as a permissive or exciting influence, we are at present ignorant; but unless we are to recast the whole of our beliefs as to the mode of nervous action, both physiological and pathological, we must accept the doctrine that each centre has this multiplicity and contrariety of function.

M. Ribot seeks the explanation of voluntary arrest in other quarters. He finds first that certain mental states—fear and pain for instance—have a primarily depressing effect. That is to say, that the occurrence of these feelings is always and of necessity accompanied by a diminution, or even an abolition for the time being, of voluntary action. Then he goes on to show that in the process of education voluntary movements are counteracted and suppressed by inducing pain or fear in the pupil—by threats and reproofs. By the law of association, the recurrence of the desire to perform the forbidden act will of itself arouse the memory of the reproofs and the threats, will induce a repetition of the accompanying state of depression, and hence will provide its own antidote. Doubtless the hypothesis is correct, so far as it attributes the suppression of the movements to the preponderance of counteracting motives aroused by the law of association; but that this counter-influence is a recurrence of the depression, and that it is the general depression of nerve discharge that prevents the occurrence of the movement, is a supposition that does not appear to be borne out by facts. Such an hypothesis would reduce the restraining influence of duty and honour to the level of mere paralysing agencies, a conclusion that would be not only ethically disheartening, but opposed to the daily experience of all men. If the stern check of duty is in the last resort a mere depression of heart and brain action, rendering us powerless to act wrongly, whence the feeling of strife, of effort, of strenuous conflict that accompanies the resolve not to do this forbidden thing? No; on the strictest adherence to evolutionary doctrine, as well as on grounds of common experience, the acquisition of the power of voluntarily inhibiting an act or a set of acts must be looked on as expressing the formation of a new stratum in the nervous centres, the addition of a new and superior rank to their hierarchy, the superposition of a new plexus, which has by virtue of its position and connections the power of inhibiting, as well as of arousing, the action of all that are subordinate to it.

When M. Ribot speaks of the will as the reaction of the entire individual upon the entire group of circumstances that affect him, all who have studied the subject will probably agree with him, and when we remember that as we ascend the hierarchy of nervous centres, each rank that we reach represents a larger and larger portion of the organism, until the highest centres of all represent the whole organism most completely, it is easy to see that from a physiological point of view the action of these highest centres is equivalent to the reaction of the entire organism.

Having laid his foundation in the description of the normal Will, M. Ribot applies the principles therein educed to the consideration of the disorders to which volition is liable. While it is not possible, nor, in so complicated, abstruse and difficult a subject, is it to be expected, that his doctrines will always meet with the reader's assent, yet every one of them is felt to be worthy of careful consideration, many of them are novel and ingenious, and all are founded on the firm base of the principle of evolution. The author's method is thoroughly scientific, and his opinions, if they do not always command our assent, invariably compel a respectful and attentive consideration. CHARLES MERCIER.

Etude Critique et Clinique de la doctrine des Localisations Motrices dans l'écorce des hémisphères cérébraux de l'homme. Par M.M, les Profs. CHARCOT et PITRES. (Reprinted from the 'Revue de Médecine'). 1 vol. 8mo., pp. 120. Paris: Alcan, 1883.

THE authors in a series of articles originally contributed to the 'Revue de Médecine' have furnished an interesting and valuable research on the subject of cerebral localisation. The basis of this inquiry is an analysis of one hundred and eighty-five cases of brain disease which have been carefully reported during life, in comparatively recent times, and in which opportunities were given of demonstrating the pathological condition after death. The general result of these observations is that there is no well-authenticated instance of a destructive lesion outside the cortical motor centres causing permanent paralysis; and on the other hand, that there is no rigorous observation of a destructive lesion of this motor zone which has failed to produce paralysis on the opposite side of the body. In short, they maintain that the facts opposed to the doctrine of central motor localisation have not been established.

The conclusions arrived at as a result of the entire enquiry are as follows:—

1. The cortex of the cerebral hemispheres in man may be divided, functionally, into two parts: motor and non-motor, according as destructive lesions do or do not cause permanent paralysis of the opposite side of the body.

2. The *non-motor zone* comprehends: *a.* All the pre-frontal region of the brain (orbital lobe, 1st, 2nd, and 3rd frontal convolutions). *b.* All the occipito-parietal region (occipital lobe, superior and inferior parietal lobules). *c.* All the temporo-sphenoidal lobe.

3. The *motor zone* includes only the ascending frontal and ascending parietal convolutions and the paracentral lobule.

4. The paralyses produced by destructive lesions of the motor zone have different clinical forms according to the seat and extent of the lesion. Thus, complete hemiplegias of cortical origin are produced by diffused lesions of the ascending convolutions; partial

paralyses are produced by circumscribed lesions of these same convolutions. The localisation of the lesion in some of these partial paralyses or monoplegias can be determined. MM. Charcot and Pitres announce the following as data for such determination: *a.* The brachio-facial monoplegias coincide with lesions in the inferior half of the ascending convolutions. *b.* The brachio-crural monoplegias coincide with lesions in the upper half of the ascending convolutions. *c.* The facial and lingual monoplegias depend upon very circumscribed lesions of the lower extremity of the ascending convolutions, particularly of the ascending frontal. *d.* The brachial monoplegias depend upon a very limited lesion of the motor zone: particularly of the middle third of the ascending frontal. *e.* The crural monoplegias depend upon very limited lesions of the paracentral lobule.

5. Whether those paralyses caused by destructive lesions be general or limited, they will in time be followed by secondary contractures of the paralysed muscles, with descending degenerations of the voluntary pyramidal tracts.

6. Irritative lesions of the cortex can give rise to epileptiform convolutions (partial or Jacksonian epilepsy), which are different from those of true epilepsy. They begin with a motor aura, and may be either generalised, or remain limited to one side of the body or one group of muscles.

7. In general the irritative lesions which cause epileptiform convulsions are localised in or near those centres which, if destroyed, would cause paralysis in the muscles affected by the convulsion. But these lesions may be even in the non-motor zone, and their relation to the functionally affected centres is not so close as is the case with paralyses and destructive lesions.

The study of the cases of monoplegia of the face, arm, or leg is particularly instructive. The authors give several figures representing the accurately circumscribed lesions which give rise to these localised paralyses. In presence of the accumulated proofs of clinical observation on the one hand, and of pathological and developmental anatomy on the other, all pointing to the same conclusions, viz., to the separation of the sensory from the motor spheres, and to the independence of the several motor centres in the human brain, the physician may look with composure upon the contradictory results of the physiological laboratory. It is quite admissible to believe that the functions of an organ of such gradual evolution in the animal series as the cerebral cortex may, according to the law of differentiation, be far more strictly localised in man than in the lower animals; and in such a wider synthesis, perhaps, the results of experimentation and clinical observation will be found to be reconcileable, nay to support one another, and we may thus hope to learn not only how things are, but also how they came to be.

A. H. BENNETT, M.D.

A Statistical Inquiry into the Nature and Treatment of Epilepsy. By A. Hughes Bennett, M.D.—H. K. Lewis, London, 1884.

This little book consists of three papers, which have already appeared in the Medical Journals at different dates during the past few years. They are now republished together, so as to form a connected inquiry. They constitute a careful and elaborate statistical investigation of many points in the clinical history of Epilepsy, and more especially of the effects of the bromides in the treatment of that disease. The author in his researches is actuated by the opinion, that the science and art of medicine is to be advanced by the collection of well-recorded facts, rather than by statements and unsupported assertions; and however laborious this may be to the physician and unattractive to the student, he believes that it is the only means by which sound therapeutic truths are to be arrived at.

The first chapter deals with a statistical investigation into the Etiology and Symptomatology of Epilepsy, and the percentage occurrences of many points connected with these conditions are given in detail. The relative frequency of the various predisposing and exciting causes in a given number of cases, as well as many of the chief symptoms and their phenomena, are demonstrated in tabular form, in such a way as to be easily accessible for reference.

The second chapter is an important contribution towards the exact estimation of the effects of the bromides on epileptic attacks. Here an attempt is made to determine, not the curative properties of the remedy on the disease itself, but only its actions on the chief symptoms, the paroxysmal seizures. This is carried out in a most systematic manner after the following plan, which shows very clearly the results of the remedy, as far as statistics are capable of demonstrating so complex a subject. From a long series of unselected cases only those were utilised for the present inquiry who had been continuously under treatment for a minimum period of six months, to a maximum of four years. In such instances all the facts of the case were carefully recorded, and more especially the average monthly number of attacks prior to treatment was determined. The drugs were then administered and steadily persevered in, and in the same way the number of attacks noted. In this manner the particulars of 117 cases have been collected and arranged in tabular form, the accumulating and recording of which represents a great amount of patience and labour. Further investigations were instituted to ascertain whether the effects of the bromides were influenced by any special peculiarities connected with the disease, such as its form, its period of occurrence, its hereditary nature, the age at which it appears, the chronicity of the disorder, the state of health of the sufferer, and so on. In all these conditions special researches have been made, and tables drawn up to indicate the results of the treatment in each. The general results arrived at are as follows: 1. In 12·1 per cent. of epileptics the attacks were completely arrested during the whole period of treatment by the bromides. 2. In 83·3 per cent. the attacks were greatly diminished, both in number and severity. 3. In 2·3 per cent. the treatment had no apparent effect. 4. In 2·3 per cent. the number of attacks was augmented during the period of treatment. 5. The form of the disease, whether it was inherited or not, whether complicated or not, recent or chronic, in the young or in the old, in healthy or diseased persons,—appeared in no way to influence treatment, the success being nearly in the same ratio under all these conditions.

The third chapter attempts to investigate the effects of the prolonged administration of the bromides on the epileptic patient, with the view more especially of ascertaining, whether in modifying the number of attacks, they in any way injuriously influence the health of the patient. The data upon which conclusions are founded consist of 141 cases, continuously under the influence of the bromides for a minimum period of twelve months, and extending to a maximum of six years. The dose of the drug was from one and a half drachms daily, upwards. These observations are also the result of an arduous and prolonged experience. The general results obtained are that, while the bromides arrest or modify the attacks, as already indicated, they, contrary to the usual belief, do not in the majority of cases seem injuriously to affect the mental or physical well-being of the patient. Tables are drawn up to show the approximative results, more especially on the physical and mental powers, on the general nervous system, and on the production of cutaneous affections. The details are given of four interesting cases, who for no less than six years have regularly consumed daily one and a half drachms of the bromide. In all of these the patients were in good physical and mental condition, and able to earn their livings by their respective occupations. The facts further seem to indicate that the symptoms of bromism diminish in intensity, the longer the remedy is persisted in. They also seem to suggest that the epileptic is more tolerant of the drug than an ordinary individual not suffering from that disease. As to whether the treatment finally eradicates the disease, there is no positive evidence to show, but all the facts lead to the conclusion, that if it was systematically carried out for a sufficiently long period, this is not impossible.

Finally, the author throughout recognises the extreme difficulty of interpreting facts—estimating results, as well as the many possible fallacies that beset the path of the investigator. While the conclusions, for the sake of convenience, have been arranged in tabular form, it is admitted that these are only approximative, and cannot be calculated with mathematical precision. At the same time the details of the inquiry have been collected with such care, and recorded with such accuracy, that it is believed they represent in the main the true facts of the case. We therefore consider that the present work of Dr. Bennett constitutes a substantial contribution to what is a very important subject, and hope his example will find numerous imitators; for nothing tends to cast discredit upon therapeutics so much as premature conclusions from observations both too few in number and too superficially performed, and the hasty publication of isolated cases alleged to have been "cured" by some drug or other.

A. DE W.

"*Text-book of the Principles of Physics.*" By ALFRED DANIELL, M.A., Lecturer on Physics in the School of Medicine, Edinburgh. (1 vol. 8vo; pp. 653. London: Macmillan & Co.)

THE task of physiology and pathology is double. First we must define and localise the function, or symptom (used in the sense of disturbed function). Second, and this is the truly scientific aspect of the problem, we must explain the function, and reduce it to its component factors. These factors are of a physical and of a chemical order; they are forces at work under conditions, the complexity of which grows with the evolution of the tissues of which they are properties. Whether there is a special force or a special modification of the physico-chemical forces; a vital force, directing the developmental and evolutionary *nisus* of organisms under varying conditions and surroundings, is a question still unanswered, at least for those whose minds are not fully satisfied by current theories. But whatever view be the correct one, physiology and pathology have essentially to do with force; the description of the mechanisms upon which the manifestations of force, the storage, liberation and transformations of energy, depend, is a mere subsidiary operation preliminary to the discovery of the laws which govern the phenomena of force manifested as life.

Now the method of studying nature is to proceed from the simple to the complex; and as in mathematics it would be idle for one to attempt to solve the problem of integral calculus who had not previously mastered the principles of arithmetic and simple algebra, so it is hopeless for one ignorant of those conceptions of force which form the basis of physics and chemistry, to launch into disquisitions concerning nerve-force and the other manifestations of energy in living organisms. Elementary as this principle seems, yet our medical literature affords ample proofs that it is very generally discarded. There is a natural tendency in the human mind to lend objectivity to its concepts; it is the most ignorant who are the most prone to give explanations because they are unconscious of the ridiculous inadequacy of their data for the solution of problems, the depth of which they cannot even distinctly appreciate.

The study of modern physics forms an essential propædeutic to that of biology. Treatises on the subject have ceased to be mere collections of experimental facts, regarding heat, light, electricity, &c., without apparent connection between them; their object is, as Mr. Daniell says, to teach the whole of Natural Philosophy as substantially a single science, in which scattered facts are connected and co-ordinated by reference to the principles of dynamics and the great experimental law of the conservation of energy. The distinction, too, between physics and chemistry is becoming more and more clearly one of convenience only; the phenomena with which they deal are but instances or embodiments of intimately correlated ideas, and it is in a study of this synthesis of the laws governing the simpler properties of matter that they shall find a most useful discipline who desire to penetrate more deeply into the mysteries of living organisms.

The book now before us is of considerable value from a double point of view. First, it is a very able one, and which fully fulfils the intention of its author to present a connected account of the principles of physics suitable to the preliminary training of medical men. Secondly, it is the only book of its kind in the English language; for though we have numerous treatises of the highest merit on the subject of Physics, yet they are all monographs. I do not mention the well-known books of Deschanel and Ganot, for they are translations only of works originally written on the now antiquated type of manuals of physics; and though their able English editors have spared no pains to add largely to their scientific value by supplying much new matter, these manuals lack the continuity of thought which is the distinctive characteristic of Mr. Daniell's book.

We need not enter here into any details as to the actual contents of the volume before us, which, we are glad to see, has been exceptionally well received by physicists generally. The author's position as Lecturer in a Medical School has enabled him to produce a work well adapted to the wants of those who wish to base their physiological and medical studies upon more solid foundations than those offered by the very imperfect curriculum still in vogue at many of our Schools.

A. DE W.

L'âme est la fonction du Cerveau. By EMILE FERRIÈRE. 2 vols. small 8vo, pp. 436 and 404. Paris, Germer Baillière, 1883.

THIS work consists mainly of an account of the nervous system as the physical substratum of the psychical phenomena of man and of animals; and of the various manifestations of mind under varying conditions of brain-development and brain-disturbance. It is a curious fact that, whilst the author starts with the professed intention to show the consonance of Spinoza's theories on mind or matter with the data of modern anatomy, physiology, and physiological psychology, his efforts should have resulted in producing a generally accurate and very readable summary of our present knowledge on the subject. The reference to Spinozism in the preface would lead one to expect some excursions at least in the field of the metaphysics of monism; but such is not the case. The author remains throughout a positivist indeed, never rising above the purest phenomenism. He shows the parallelism between nervous organisation and mental manifestations, and contents himself with calling the latter a *function* of the former. It would have been well if he had defined this word "function," which to us seems to explain nothing, and leave the problem—an insoluble one according to most competent thinkers—very much where it was.

When we said that the metaphysical aspect of the question has been left untouched—nay, apparently ignored—by the author, we only referred to the constructive part of his book. There runs throughout its pages a vein of controversial aggressiveness against the popular "Spiritualistic" conception (still represented in the official philosophic teaching in French Academies) of a dual nature of man, who according to this theory consists of an independent spiritual entity united to a material organism, of a spirit and a body mysteriously acting and reacting upon one another. It is possible that for the general reader this constant repetition that the psychical phenomena observed in man and the lower animals, in health and disease, are not compatible with this view, may be found advantageous or necessary; but to the student of either of the great metaphysical systems or of modern psychophysiology such an instance will appear somewhat puerile. In the course of his controversy M. Ferrière lays considerable stress upon an argument by analogy, which however appears to us more specious than solid. He tries to show by a parallel between the attributes of mind as a function of the brain, and those of magnetism as a "function" (*sic*) of the magnet, that there is less difference between these "immaterial forces" than between the material products of two glands, such as the spermatic and lactic secretions. True, magnetism disappears when the outer film of the magnet is dissolved in an acid, just as psychical manifestations do when the cortical layer of a brain is removed; but how superficial are all such analogies! Magnetism is a force correlated to heat, light and electricity, and postulates space for its manifestations; mind is subject to conditions of time only, is not a force, and does not originate in any transformation of the forces set free by that liberation of energy which constitutes nerve-discharge. Nor can mind, not being a force, ever liberate energy, and originate any of the cerebral processes which are the necessary conditions of thought, feeling, as well as of movement; it is neither a cause nor an effect of the physical changes involved in nerve action; it neither precedes nor follows, but accompanies those changes. Mind is coextensive with consciousness; if it were the product of brain action, the equivalent of a certain quantity of nervous energy which might be supposed to be converted into vibrations (of a mentiferous ether, as it has been suggested) our mental processes would be uniformly proportional to the quantity of nervous matter oxidised: but this is not the case, and identical cerebral acts differ largely as to the intensity of the conscious states which accompany them. In other words "cerebration" and "mentation" do not both obey laws reducible to those of transformation of energy. There is no common term between the reflex process in which a nerve excitation from the sensory sphere liberates in a motor centre a certain quantity of stored-up force, which in its turn sets free energy in certain groups of muscles, and the mental state we call a volition.

M. Ferrière, like many students of neurophysiology, has been so dazzled with the apparent simplicity of the method, now so popular, of expressing psychical in terms of physical phenomena—mind in terms of matter—that he has lost sight of the very problem which he gave himself the task to solve. The fact is that we must either remain content to leave the answer in the domain of the unknowable, or launch into the abyss of metaphysics. Mind, in Spinoza's own language, is the idea, or, as we should now call it, the consciousness, of the body; not a "substance" but a "mode"—a collection of states. Body is the subject of the soul; both are modifications of one substance with attributes infinite in number, but cognisable to us under two only, matter and thought. Hence mind and body are but one phenomenon under two aspects—the same ray of divine light, refracted as it were, and analysed through the prism of psychological introspection. Even from the standpoint of our modern positive science many will feel more sympathy for the bold speculations of the great metaphysician, than for the so-called materialistic solution of the problem which consists in suppressing one of its terms.

This book is very readable, and full of information on every topic connected with the nervous system considered as the physical basis of mental manifestations. The style is very clear and the facts well arranged. The chapters on the anatomy and embryogeny of the nerve centres, however, might have been advantageously either curtailed or illustrated.

A. DE W.

Neurypnologie; Traité du sommeil nerveux, ou hypnotisme. By JAMES BRAID. Translated into French by Dr. J. SIMON; with a preface by Prof. BROWN-SÉQUARD. 1 vol. small 8vo, pp. 268. Paris: Delahaye, 1883.

THE study of the phenomena of somnambulism, or hypnotism, has during the last few years been prosecuted with much energy and success, and we are glad to find the suggestive work of Braid placed before continental readers in the shape of an excellent French translation. This volume will be welcome in England also, both on account of the scarcity of Braid's original edition

(1834), and of the new additional chapter which it contains. This chapter consists of a communication made by Braid in 1860 to the Academy of Sciences, in the very month of his death, and occupies thirty-five pages in the translation.

The "Neurypnology" is divided into two parts. The first, historical, experimental and controversial, contains an *exposé* of Braid's methods and theory. His merit consists in having exploded the "animal magnetism" craze, and shown that the hypnotic condition is induced by sensorial impression of various descriptions. Instead of the classical "passes" made over the patient by the operator's hands, he used the convergence of the eyes inwards and upwards, such as in fixation of a small object placed near and above the place of ordinary vision.

This portion of Braid's work is too well known for us to say any more about it here. The second part is therapeutical, and though at the time of publication it aroused a considerable amount of ill-feeling against the author, it has now passed into comparative oblivion. But the time is near when the curative influence of hypnotism will be submitted to the same scrutiny as its physiological and psychological import has undergone. There is certainly much exaggeration and self-deception in Braid's enthusiastic report of the cases he declares to have been successfully treated by himself. Still there is no doubt a substratum of truth in what he says; and we have no right to refuse *à priori* to believe an observer of such undoubted uprightness when he begins to speak of the practical applications of a discovery in the elaboration of which he has shown true scientific insight.

The cases in which hypnotism appears to have relieved the symptoms more or less permanently belong chiefly to the group of neurotic functional disturbance; the method consists simply in hypnotising the patient (several times if necessary) and allowing him to recover naturally from the somnambulic state. A few well-observed instances in which this has been efficacious have been quite recently published in Germany, where Profs. Preyer and Heidenhain's physiological researches on hypnotism have excited a considerable interest. Thus Dr. Wiehe ('Berliner Klin. Wochenschrift,' January 1884) experimented upon four patients in Prof. Bäumler's Clinique at Freiburg. Two cases (of hysterical nature), in which persisting and violent clonic spasms formed the chief symptoms, were the one relieved, the other cured of the complaint. A third patient was freed of hemi-anæsthesia; and the fourth, who had been vainly submitted to electrical and other treatment for neuralgic pains in the face and arm, was completely relieved of the symptoms by one hypnotisation. In other cases of hysterical disturbance, however, hypnotism failed. It is not to be forgotten in experiments of this kind that a repetition of the operation may prove injurious to the nervous system—for instance, leave a tendency to spontaneous somnambulism in predisposed subjects. Hypnotism may in certain cases prove a valuable substitute for the usual method of inducing anæsthesia; Braid and others have reported instances where surgical operations have been carried out painlessly on hypnotised subjects. But whatever be the practical value of hypnotism from a medical point of view, the light it has already thrown upon the mechanism of cerebral action, and the important place it must henceforth occupy in experimental psychology, are enough to encourage fresh efforts in the investigation of its phenomena. The publication of the "Neurypnology" is a well-timed homage to the memory of a man who, with singular tenacity of purpose and true singleness of heart, incurred much obloquy and persecution in the pursuit of science and truth.

A. DE W.

Medicinische Bibliographie. By DR. ARTHUR WURZBURG, Librarian to the Imperial Sanitary Office in Berlin. Vol. I. 1883, 8vo, pp. 386. Leipzig: Breitkopf and Härtel.

THIS publication will be found extremely convenient by everyone who wishes to be kept *au courant* of the ever-increasing literature of the medical sciences. For the modest sum of six shillings the subscriber receives weekly a part of six to eight pages, in which all the recent works and papers of importance are indicated by title and reference. They are arranged under about twenty-five headings, one of which, that of nervous diseases and psychiatry, will prove of special interest to our reader, and keep him informed of the latest papers to which he may have to refer.

The size of this 'Bibliographie' excludes, of course, the possibility of its pretending to the same exhaustive completeness as the American 'Index Medicus,' now defunct, we fear, for want of support by the profession at large. It will nevertheless be well received, as it thoroughly fulfils the eminently practical design of its able Editor.

A. DE W.

Clinical Figures. Brain: I. after ECKER; II. after PITRES. Lebon & Co., 28, Southampton Buildings, Chancery Lane, London.

THESE figures are published on sheets of paper, gummed at the back, so as to be readily transferred into a note-book. Each sheet contains six figures, and there are twenty-five sheets in a book. The first series comprises views of the surface of hemispheres (superior and inferior, right and left external, right and left internal aspects); the second reproduces the very useful sections of Pitres. This publication fills a long-felt want. They will be found invaluable in the post-mortem room; also exceedingly convenient to the student of cerebral pathology, who is thus enabled to take rapid sketches of the interesting localisations of brain-disease with which he meets in the course of his reading.

Messrs. Lebon have conferred a further boon upon neurologists by announcing that they will supply the electroplate-block of any of the twelve figures, with the outline of the lesions which it is desired to illustrate. All that the author has to do is to indicate on one of the diagrams the position of the hæmorrhage, tumour, &c. His markings are transferred upon the cliché, the printed figure from which shows the lesion as a lighter patch upon the darker ground. The cost of illustrating cases of brain disease is thus reduced to a very trifling sum (about two shillings for each figure).

Messrs. Lebon are preparing a similar series of diagrams of the spinal cord. Other series have been published, illustrating the eye and retina, the surface of the head, of the body, &c., all of which will occasionally be found useful for a graphical record of the symptoms observed in cases of nervous disease.

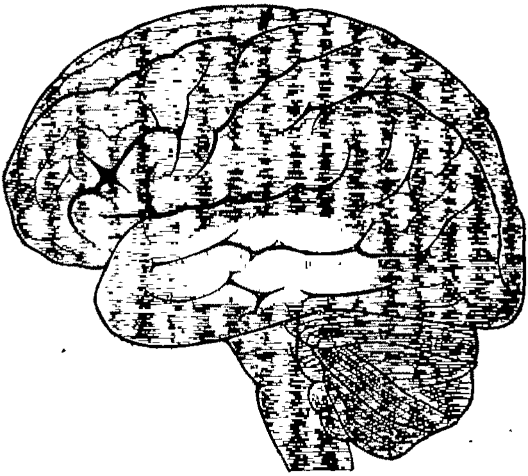

FIG. 1.

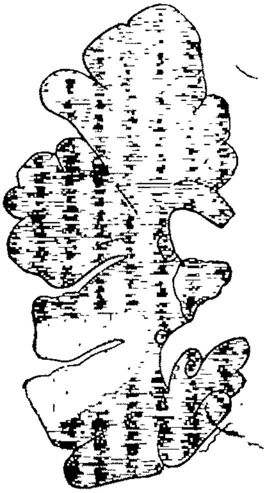

FIG. 2.

As specimens of the clinical figures of the brain, and to show the way in which localised lesions may be illustrated by their means, I have appended two figures illustrating a patch of softening in the left temporal lobe. Figure 1 depicts the superficial extent of the same; Fig. 2, its depth on a pediculo-parietal section of the brain. The case from which they are taken is one of *verbal deafness* recently published by Prof. Tamburini, and which will be referred to more fully in a future number of 'BRAIN.'

A. DE W.

I. J. ROSENTHAL *and* M. BERNHARDT: *Electricitätslehre für Mediciner und Electrotherapie.* Berlin: Hirschwald, 1883. 521 pp.

II. S. TH. STEIN: *Die Allgemeine Electrisation des menschlichen Körpers.* Zweite Auflage. Halle: Wilhelm Knapp, 1883. 144 pp.

I. THE first part of this work (12 chapters) is the third edition of Rosenthal's well-known book on Medical Electricity, carefully revised and adapted to the present standard of this discipline. For English students it is perhaps a little too thorough, in so far as the author has not limited himself to those facts which the practitioner requires to be acquainted with before he can make a practical use of electro-therapeutics, but he also gives an account of facts and methods which are used in physiological research. This, however, can hardly be considered as a deficiency of the book, for the author treats the subject with much conciseness and remarkable perspicuity of style.

The second part is Professor Bernhardt's own work. It contains 16 chapters on the application of Electricity to the diagnosis and treatment of diseases. Two chapters are devoted to remarks on batteries, galvanometers and other apparatus, and to an account of the motor points and the methods of investigations of the motor nerves and muscles. Then follows a description of the pathological alterations of electric irritability, and of the therapeutical effects of Electricity in general. The greater share of this part of the book belongs to the description of the therapeutical application of Electricity. An Appendix on Metalloscopy, Metallotherapy, and static Electricity, and a number of interesting clinical observations, chiefly illustrating the use of electro-diagnosis, completes the book.

It is hardly necessary to say that Professor Bernhardt, whose works represent an essential part of electro-therapeutical literature, has fulfilled his task in a masterly style. The book is free from merely hypothetical reasoning; it deserves to be carefully studied by all those who take an interest in the diagnosis and treatment of diseases of the nervous system, for which purpose a thorough knowledge of the medical applications of Electricity is absolutely indispensable.

II. The fact that the first edition of this little book has been sold out within six months is a striking proof of its practical value. The chief object of it is to show the use of Electricity applied to the entire surface of the human body (or the greater part of it), especially in cases of neurasthenia and similar affections. This kind of treatment was first recommended by Beard and Rockwell, of New York; but while the American authors exclusively applied the faradic and galvanic currents, Dr. Stein also advocates the use of static electricity or Franklinisation. He refers to Charcot's and Vigouroux's experience, to which he adds a number of observations of his own. The electric bath, too, is extensively described and much recommended. Besides this, the little book contains many useful hints concerning the choice and handling of electric apparatus; it is in every way an interesting publication, even for those readers who may not be able entirely to agree with the author's views and expectations.

R. H. Pierson, M.D. (Dresden).

Abstracts of British and Foreign Journals.

Hughlings-Jackson on Evolution and Dissolution of the Nervous System.—(*Croomian Lectures for* 1884. *Brit. Med. Journal*, March 29th, *ff.*)—The Croomian Lectures were delivered in March of this year at the Royal College of Physicians by Dr. Hughlings-Jackson, who chose for his subject the "Evolution and Dissolution of the Nervous System," and gave what may be regarded as a highly condensed summary of the results of the work which has occupied him for many years. At frequent intervals Dr. Jackson has published, in somewhat fragmentary form, the results that he has arrived at in localised districts of his field of work; but these publications have been distributed over so long a period, and disseminated in so many journals, that they are not readily available for reference. These lectures are therefore welcome as a recapitulation in an accessible form of much that has appeared before; as giving the latest outcome of Dr. Jackson's labours; and as presenting his views in a more unified, or as he himself might term it, a more integrated shape than they have yet assumed. Dr. Jackson regards the central nervous system as a hierarchy; in which each grade controls the grade below and is controlled by the grade above; each grade represents over again and co-ordinates in more elaborate combinations the parts represented and co-ordinated by the grade below; and every part or region or centre of a grade represents a larger share of the organism than any corresponding part of the grade below, a more limited share than any corresponding part of the grade above. Thus each centre in the lowest grade represents but a limited portion of the organism, and each centre in the highest grade represents the whole of the organism, but in no two of the latter centres is the whole organism represented in quite the same way. The whole of the grades are grouped in three main divisions—lowest, middle, and highest centres. Of these the lowest are the most completely organised, the highest the least completely organised. When disease attacks the nervous system, there are always two sets of manifestations: negative and positive—loss of function and excess of function. The functions lost are those of the centres diseased. The functions that are excessive are those of centres subordinate to the centres diseased, and permitted to act excessively by the removal of the control normally exercised by the centres now diseased. The centre whose function is abolished may belong to the lowest, to the middle, or to the highest division, affording examples of local dissolution. On the other hand there may be a uniform dissolution, the whole of the highest grade of all being first lost, and successive grades being pared off, as it were, as if in layers. Dr. Jackson applies these doctrines, with many subsidiary hypotheses, to every kind of nervous disorder, from atrophy of muscles to insanity, and from giddiness to coma; and from his vast knowledge of diseases of the nervous system he is able to supply an apparently limitless profusion of instances to every section and subsection of his subject. C. MERCIER.

Ballet on Exophthalmic Goitre. (*Revue de Médecine*, April 1883.)—The general results at which the author arrives after an inquiry into this subject are as follows:—

1. To the classical symptoms of exophthalmic goitre (palpitations, swelling of the neck, tremor,) there are occasionally added others which, like them, are attributable to disorders of the nervous system.

2. These symptoms are on the one hand *convulsive* (epileptic or epileptiform attacks), or on the other *paralytic* (hemiplegia or paraplegia); also pretty frequently albuminuria, glycosuria, or simple polyuria may be observed.

3. These convulsive or paralytic complications appear to arise, not as the direct result of the Basedow's disease, but of another coincident neurosis (epilepsy, hysteria).

4. Sometimes certain convulsive phenomena (epileptiform attacks) seem intimately associated with exophthalmic goitre itself, and the special clinical conditions under which they are manifested authorise us in connecting them with disturbance of the cerebro-bulbar circulation, itself occasioned by perturbation of the action of the heart.

5. Among the paralytic disturbances some are of minor importance such as the weakness of the hands, the temporary paresis of one or both upper extremities, and the feebleness of the lower limbs. They may be looked upon as directly dependent either upon the tremor, or on functional derangement of the cerebral circulation.

6. The polyuria, albuminuria, and glycosuria are probably more frequent than might be supposed from previous researches, and they indicate a derangement of the bulbar innervation.

A. H. BENNETT.

Dr. Franz Zimmerlin on Hereditary (family) progressive muscular Atrophy. (*Zeitschr. für Klin. Med.* Band 7, Heft 1.) —The clinical picture of hereditary ataxy has been classically worked out by Friedreich; the hereditary form of progressive muscular atrophy by Leyden. The hereditary form of progressive muscular atrophy, in opposition to the not hereditary, is classified as a clinical secondary form by Leyden, as follows:—

Beginning of illness nearly always in advanced childhood, seldom later, attacks several members of same family, particularly males. Begins with weakness and atrophy in loins and lower extremities, first in lower part of leg or muscles of back; it is not always recognizable, owing to development of fat in the interstitial tissue. After some years, shoulders and arms are attacked; the change is one of pure atrophy, *i.e.* not complicated with fatty changes.

The following cases show that not only one, but at least two types of this etiological secondary form exist.

Zimmerlin gives seven cases, belonging to two families: four in one family (Loosli, Bern), and three brothers (Schuhmacher, Frickthal).

In the Loosli family ten children are living out of eleven. Two eldest, 28 and 30, are strong and healthy, also three youngest, 14–19. Of five between 20 and 26, one brother, 24, quite healthy. The other four, two brothers, 26 and 22, and two sisters, 25 and 20, are affected, and show severe disturbance of nutrition and function of voluntary muscles.

The father has pulmonary phthisis, mother quite healthy. No neurotic heredity. Family intelligent and well-built.

As an example, Verena Loosli, is 20 years old. Childhood weakly, with croupous pneumonia; at puberty she had erythema nodosum. Probable commencement of illness in summer 1881, with weakness in the back, fatigue in standing or sitting upright. In autumn, pain in region of right shoulder-blade, fatigue in both legs, with slight formication.

Since January, 1882, patient remarked that her shoulder-blades gradually began to come forwards, and the posterior borders projected from the chest-wall, chest flattened, clavicles more prominent, at the same time she had some weakness in the upper extremities; could not lift up her arm to her head, but could raise arms to the horizontal position by means of the deltoids. The spine, at the sixth dorsal vertebra, was slightly painful on pressure. Atrophy and paralysis in a high degree of both serrat.

antic. magn., with complete loss of excitability of muscles and their nerves for galvanic and faradic currents. Decided weakness and degeneration-reaction of both pectoral major; moderate but distinct weakness and commencing degeneration-reaction of acromial part of right trapezius; lessened capability, atrophy and degeneration-reaction of right and left biceps brachii. Weakness but not atrophy in flexors of right elbow. No wasting of deltoids, of the small muscles of hands, or interossei, no fibrillar tremor. Sensibility of skin normal.

Treatment consisted of methodical galvanisation of cervical spine, galvanisation and faradisation of affected muscles, moderate hydropathic course, and it produced some slight improvement.

Of the other members of the family, in two brothers aged 26 and 22, the disease began at 21 and 18 years respectively, while in a sister, aged 25, the first symptoms were noticed at the age of 23. One brother had a slight systolic apex murmur, and slight pulmonary phthisis. The disease had the same commencement, and the same general characters as in the case described.

Summary.—Identical points in all four cases are:

Beginning of illness soon after puberty, between 18 and 23. Localisation in upper part of body (especially upper half of trunk and upper extremities, whilst lower part of body was almost entirely free from disease), predilection for certain larger muscles (serrat. antic. magn., pectoralis, biceps, triceps brachii, extensors and supinators of forearm of both sides of body), immunity of muscles of thenar and hypo-thenar, interossei and deltoids, absence of fibrillar tremor in atrophied muscles, absence of secondary "lipomatosis luxurians;" no particular disturbance of sensibility, or at least decided absence of hyperæsthesia and anæsthesia. The disease presents itself in all four cases in a gradually increasing atrophy and weakness of the muscles mentioned, and deserves the name of "progressive muscle atrophy," inasmuch as it, successively attacked different muscles, which gradually progressed to the highest degree of atrophy. It agrees further with "progressive muscular atrophy," in so far as (at least in Case I.) the faradic and galvanic investigation of the attacked muscles and their accessory motor nerves has produced characteristic results (excitability of muscles and nerve retained for both currents, as long as the atrophy of the muscles had not become extreme; degeneration-reaction, to a moderate degree, of the individual atrophied muscles).

The disease differs from progressive muscular atrophy, in the hereditary disposition, and in not affecting the small muscles of hand and interossei, also in the absence of fibrillar tremor. It agrees with Erb's progressive muscular atrophy of youth, which affects the larger muscles of shoulder and arm, lasts for some decades, and then remains stationary and presents no fibrillar tremor; but differs in the fact that, in Erb's cases, the large muscles of thigh and buttocks are attacked; there is no electric reaction of degeneration and no family predisposition. The described cases agree with one of Schulze's in most points, but in his case hereditary disposition is absent. They differ from the cases of Leyden and Moebius, where the lower extremities are affected, no "lipomotosis luxurians" is present, the male sex chiefly attacked, and childhood is affected first.

Zimmerlin refers to another family, named Schuhmacher, in which three brothers were affected; no hereditary neurosis, no insanity; family consisted of thirteen children; seven died young, cause unknown; three have atrophic paralysis. The disease began at 15, 13, and 8 years old respectively, with weakness and atrophy of both serrat. magn., rhomboids, pectoralis major of both sides, and to a less degree, biceps, triceps, supinators and extensors of forearm; while the deltoids, trapezius, infra- and supra-spinati, and small muscles of hand are not affected, and no fibrillar tremor. Differs from the Loosli family in all being boys, and so resembles Leyden's cases. In one case the mouth was a little drawn to the left, and he could not whistle. Some atrophy of left biceps, and to a less degree of left triceps, but their tendon reflexes are present; some atrophy and weakness of supinators and extensors of wrists; extension and abduction of right thumb weak. In the third case, which has existed for thirty-four years, both arms are wasted and weak, even the interossei and small muscles of hand; both thighs affected and almost powerless, extensors worse than flexors; both knees contracted, arthritis deformans in the right; no patellar tendon reflex; no "lipomatosis," no fibrillar tremor now. This last case began first in lower limbs; afterwards affecting the upper limbs, and fibrillar tremor was observed at first.

Dr. Zimmerlin thinks the disease in the two families shows an inherited tendency. In the Loosli family, severe muscular work may have been a cause, and the patients thought the disease came on at the time of life when they worked hardest.

He thinks that three possible pathological causes of the disease exist: 1. Myopathic change, primary disease of the muscles;

2. Neuropathic condition, multiple neuritis (Leyden); 3. Localisation in spinal cord, as a form of chronic poliomyelitis anterior. The second condition is negatived by absence of anæsthesia, and of marked electric changes. An autopsy can alone prove whether the disease is in the muscles or the spinal cord.

C. E. Beevor, M.D.

Dr. C. Eisenlohr (*Neurolog. Centralbl.* Feb. 15, 1884) describes a case of chronic spinal meningitis, with secondary degeneration of the spinal cord, probably of syphilitic origin.

Among the most important symptoms were paralysis and loss of sensation of the lower extremities, both more marked on the left side, with rigidity of the right leg. Also very obstinate constipation, incontinence of urine, and dull boring pains in both legs and in the rectum. History of syphilis eleven years ago. Present symptoms date from about one year ago. Temporary improvement under brisk mercurial treatment. Appearance of bedsores over the sacrum and great trochanters. Loss of sensibility confined to posterior and outer aspect of the thighs and calves, fairly good on anterior aspect. Hyperalgesia of dorsum of both feet. Plantar reflex weak on the right, absent in the left. Right leg much wasted. Patient died nearly two years after admission.

Post-mortem—was found great thickening of the pia mater, which was adherent to the dura, from the middle of the cauda equina to the level of the 10th dorsal nerve. Nerves of cauda soldered together. The thickening extended in a less degree to the mid-dorsal region. Nerve-fibres of cauda much degenerated. So also are the posterior roots of the whole lumbar region, more particularly on the right side, anterior roots having escaped.

Cord.—Lower sacral lumbar region, whole posterior columns quite degenerated. Higher up, however, the degenerated area becomes smaller and confined to the median column. The degeneration can be traced up into the cervical region affecting a diminishing area of the postero-median columns, till it is lost in the nuclei of the funiculi graciles. The fibres of the right sciatic nerve are greatly degenerated.

The author remarks that this case adds testimony to the views already expressed by Kahler and Pick, that the so-called Goll's columns are not distinct topographically from the neighbouring region, but are simply the longest centripetal continuations of the posterior roots, in this case supplying the lower extremities.

H. H. Tooth.

BRAIN.

OCTOBER, 1884.

Original Articles.

THE CEREBRAL BLOOD-VESSELS IN HEALTH AND DISEASE.

BY PROF. H. OBERSTEINER (VIENNA).

THE great progress which microscopical science has made in the last ten years,—in which I include the method of hardening, preserving, staining, &c., as also the improvement in the optical part of instruments,—has taught us not only to know more about the finer structures of animal tissues and their elements, but has also revealed an increasing number of changes, which denote the impress of a pathological condition.

Latterly certain sceptical and unprejudiced investigators have undertaken to verify more exactly many of these so-called pathological changes in the histological structure of the tissues, to determine whether we really have to do with abnormal morbid conditions.

The conditions found are as follows :—

1. We have a true pathological tissue change; or

2. We have to deal with changes due to the methods of preparation (decomposition, hardening, &c.); or

3. The apparently morbid change is frequently found in tissues quite healthy; or

4. Lastly, we may have to do with histological conditions which are met with so constantly in normal organs, that in a way they may be regarded as characteristic of healthy structure.

I consider it most essential to have some test of the true value of the appearances seen by the microscope; the immense array of material which has been collected must be properly arranged, and many errors must be discarded.

I have, some time back,[1] occupied myself with the relations of the small vessels of the brain, to settle the boundary between the normal and pathological vascular changes. In spite of this, I have very frequently found that the occurrences of vessels considered to be diseased, have afterwards been found present, and never absent, in every healthy brain. It is to be noticed that such a false explanation brings in its train a series of false conclusions, and often in considerations of very great importance.

I must here explain that the above-mentioned work is not generally known to English readers, and I think it advisable to bring before them in these pages some of the points in my work, and the reader will pardon me if I occasionally repeat what has been already published.

It is scarcely necessary for me to refer to the important meaning which pertains to the blood-vessels of the central nervous system. It should be assumed that the nervous elements, and especially the ganglion cells, are more sensitive than any other parts of the tissues to disturbances of their normal nutrition. The anatomical and physiological entirety of the blood-vessels is necessary chiefly for the normal function of the organ of the central nervous system; and therefore it is again evident how very important it is to distinguish between the normal and morbid vascular changes.

In the following pages I shall not take account of the larger vessels, which run outside the brain and spinal cord; but I will confine myself to the smaller vessels, which are already imbedded in the nerve substance.

Methods of investigation.

The best representation of the structure of the smaller vessels of the brain is obtained when they are examined in the fresh condition, and with the use of as little chemical reagents as possible. I have made it the rule in every case, where a diseased brain is in question, to make examination of the fresh organ, that is of the small vessels, and I can very strongly recommend this practice; for, after hardening has occurred, nothing much can be recognised. Pieces of brain or spinal cord, not too small, must be macerated in a pale yellow solution of bichromate of potash, for 24 hours (2–4 days will not hurt). If the brain-substance be now coarsely separated numerous vessels are found, which can be isolated (always under water) by means of a couple of needles from the surrounding nerve-substance. In this way preparations can readily be made of the entire arterioles with their ramifications, together with the beginning of the capillary network; and it can be so managed that the vessel is scarcely, or not at all, damaged by the needle, if it be held firmly on the surrounding nerve-tissue.

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[1] 'Beiträge zur Pathologischen Anatomie der Gehirngefässe,' Wien. Med. Jahrbücher, 1877.

The prepared vessel can now be immediately examined in water, or very weak salt-solution, and thereby most of the true details of the structure can be recognised. If the edge of the cover glass be surrounded with damar-lac, and care be taken to do this thoroughly, the preparations can be kept for a very long time in water (I have kept some for ten years) without any appreciable change (with a few solitary exceptions).

The preparations can be stained by aqueous solutions of various dyes (carmine, picrocarmine, hæmatoxylin, aniline dyes), and after rinsing in water, they are to be preserved in the water as shown above.

The generally used fluids, glycerine, oil of cloves, &c., are to be avoided, as they produce marked changes in the vessels.

Certain pathological conditions—there are only a few—should especially be studied in cut sections; for this purpose the well-known methods of hardening are to be employed.

Structure of the normal vessels of the central nervous system.

1. *Structures of the arteries.*—If we examine the smallest arterial vessels, we can distinguish four coats, which I will

describe, from inside out, as endothelium, membrana fenestrata, tunica muscularis, and adventitial lymph-sheath.

(*a.*) The endothelium is a very delicate membrane (Fig. 1, *a*) consisting of a simple layer of flattened cells; the ground substance between the cells can be demonstrated by the usual method with nitrate of silver. The nuclei of the endothelium-cells, which become apparent after staining with carmine, are an elongated oval in shape, or like a whetstone, and are arranged with their long axes in the direction of the flow of blood. In Fig. 1, where the other layers are present over the endothelium, the cells of the latter are clearly to be made out by the longitudinal arrangement of their nuclei. Nearly always at the edge of the cell-nucleus is seen a strongly refracting bright granule, which projects into the nucleus itself, and the meaning of which is not yet explained.

The endothelium has a tendency to run into folds, when it is detached from the other coats; it can be found best (together with the membrana fenestrata) by teasing with needles one of the intra-cerebral arterioles, after they have been stained with carmine. Th. Deecke[1] has observed, within the just described endothelial flattened cells, yet another layer of cylindrical cells, and he adds further, that the innermost layer of cylindrical epithelium disappears in the smaller branches, "which have entered the brain-substance, and always after their first division."

(*b.*) *The membrana fenestrata,* Fig. 1, *b*, contains no nuclear or cellular constituents, but appears as a glassy, compact, membrane, which always presents very sharp, clear, longitudinal folds. When more highly magnified, numerous small bright spots (or holes ?) are to be seen. They are most visible in one of the large arteries, which are very numerous in the basal cerebral ganglia. In the arteries of the convexity of the brain the membrane is extremely delicate, and disappears usually as the calibre of the arterioles diminishes. One of the important layers outside the membrana fenestrata, which Deecke (loc. cit.) described as a "tendon-like membrane," I have failed to discover,—at least in the smaller arteries.

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[1] "The Structure of the Vessels of the Nervous Centres in Health, and their Changes in Disease;" 'American Journal of Insanity,' 1877–81.

(*c.*) *Tunica muscularis* (Fig. 1, *c*) consists of spindle-shaped smooth muscle-fibres, which encircle the inner layers of the blood-vessel. The sharply notched edge of the vessel gives a clear representation of the individual muscle-fibres, the long oval nuclei of which are distinctly seen after staining with carmine; they are all arranged transversely, and cross the corresponding nuclei of the endothelium. On the larger arteries are many layers of muscular tissue, placed one over the other, and these determine the thickness of the vessel-wall. The finer arteries possess only a simple layer of muscle-fibres, and moreover their form alters with the diminishing size of the arteries, in such a way that the fibres (and also their nuclei) become shorter, and at the same time broader.

Deecke describes, outside the tunica muscularis, a layer "formed by separate large bundles of elastic fibres." I had already seen these fibres, and have described them in my treatise above quoted. I have explained these, as folds of the next layer, viz. the adventitia. I confess that I have frequently entertained doubts as to the accuracy of my view, without on the other hand being able entirely to accept that of Deecke; this, however, I should by no means oppose, that these questionable fibres are found exclusively in the large arteries, and are not present in the smaller ones, a point which Deecke does not mention.

(*d.*) *The adventitia* (the adventitial lymph-sheath, Fig. 1, *d*). This is a delicate connective-tissue membrane, which, extending very much further than the tunica muscularis, is not attached to it, so that between the two coats there is a freely communicating space. The adventitia contains round or oval nuclei, which frequently have a little protoplasm around them, and in appearance they resemble connective-tissue nuclei. Situated on the adventitia are more or less pigment granules, fat-globules, and fat-granule cells; about these normal appearances I shall soon have to speak further. Concerning the presence of endothelial cells on the adventitia I am not more certain than is Deecke; on the other hand, I must endorse the opinion of this author concerning the character of the adventitia, that it is a perfectly independent sheath.

In cut sections, particularly from the brains of animals, we see sometimes very clearly, as if appearing from the adventitia, stiff, long-drawn-out processes, which project into the brain-substance, and end in the connective-tissue cells. In fresh-teased preparations there is nothing to be seen of these numerous processes; I must confess that it does not seem to me quite clear how, in preparations made as delicately as possible, all these fibres, or the greatest part, can be torn away from the adventitia.

For the most part, I incline to the opinion that, outside the adventitia, a delicate connective-tissue membrane exists, somewhat like an inner coating to the canal in which the vessel courses.[1] This outer sheath of the vessel, derived from the pia mater, is so intimately connected by the numerous above-mentioned processes with the connective tissue present in the interior of the brain-substance, that, even with the most delicate manipulation, this membrane remains behind, while the whole vessel with its adventitia is detached. In a hardened preparation the outer sheath is so closely applied to the adventitia, and this is so crumpled up with the inner vessel-coats, that it can no longer be recognised as an independent membrane.

I scarcely need affirm, that the adventitial vessel-sheath is in no way intimately fused with the brain-substance which forms the ultimate external sheath; there remains a space between the two, which frequently appears to be completely reduced to a minimum. We have outside, as well as inside, the adventitia a space where fluid can circulate; under normal conditions we cannot speak of any fluid, besides the animal juices, which are set apart to bring about the change of matter between the blood and the tissue of the organ; we can designate this juice shortly as "lymph," and from this we can speak of two different lymph-spaces, one between the tunica muscularis and adventitia (adventitial lymph space, *Virchow, Robin*), while the other is outside the adventitia (perivascular lymph-space, *His*). In both named lymph-spaces, under certain conditions, we find, more or less, numerous lymphoid corpuscles; but whether they are, as Deecke thinks, only wandering white blood-corpuscles, it is not easy to decide.

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[1] Compare Adler, 'Archiv für Psychiatrie,' 5 B.

I should like to remark, with regard to the perivascular lymph-space, that it should be looked upon as a cavity between the tissues, and that it communicates with numerous other cavities, which serve to bring about the circulation of the nutrient fluid to the tissue elements. I have shown[1] that the ganglion cells of the grey substance are situated in a hollow space, "pericellular lymph-space," which under certain conditions may be very small, but it is always present—small though it be—between the protoplasm of the nerve-cells and the neuroglia. These pericellular spaces are in communication with the perivascular spaces, and are certainly just as little an artificial product as the latter. The proofs of this can be found in both my above-quoted works, as well as in another essay.[2]

2. *Structure of the veins.*—In the intracerebral veins I can distinguish only three layers.

(*a.*) *The endothelium* is much more irregular than that of the arteries, the cell-nuclei are for the most part rounder, not so elongated and less symmetrical, and are situated in a position corresponding to the longitudinal direction of the vessel.

(*b.*) *For the middle coat,* we must take that tissue which forms the principal part of the wall of the vein; it consists of connective tissue with scattered round nuclei, and only a few irregularly arranged muscular fibres.

(*c.*) *The adventitial lymph-sheath* corresponds with the description of the arteries given above, with a single difference, about which I will speak later.

I may mention that *Deecke* gives a description of the structure of the cerebral veins, differing from mine in many points.

3. *Structure of the capillaries.*—In these, not more than two layers can be seen.

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[1] 'Ueber einige Lymphräume im Gehirne,' Sitzungsber. der K. Akad. der Wissensch. zu Wien, 61 B.

[2] 'Ueber Ektasien der Lymphgefässe des Gehirns,' Virchow's 'Archiv,' 55 B.

The capillary tube proper is formed from a continuation of the arterial and venous endothelial membranes, which have been divested of their outer coats, only the adventitial sheath being visible in many places; I hold the opinion that the adventitia forms round the arteries and veins, as well as all the intracerebral capillary system, an uninterrupted sheath; only over the capillaries this is finer and narrower, so that it often escapes observation.

Further peculiarities of the structure of the adventitial sheath of the brain-vessels.

I have mentioned earlier, that in the adventitia pigment and fat are found; on this point I must make some further observations.

When we look at the central nervous system of new-born children, we find numerous fat-granule cells; it is highly manifest that these play a considerable part in the development of the medullated fibres; they convey the material for the formation of the medullated sheath, just as, by morbid dissolution of the medullated fibres, the fat-granule cells again appear, and take up matter they come in contact with and remove it.

Similar fat-granule cells are found again in the new-born child, in the midst of the whole nervous system, and not a few are firmly attached outside the adventitia of the vessels; the flow of lymph is quite incompetent to tear them away, and so they remain like "stranded waifs," when all the other fat-granules have disappeared from the central nervous system, the development of the medullated fibres being completed.

If we now examine some of the vessels (artery or vein) from the brain of a child under two years old, we shall find, perhaps not on every vessel, still very frequently, these already-described fat-granules, which under a low magnifying power stand out very clearly as dark specks. We shall certainly find, after a short search, in every child's brain such vessels with fat-granules on the adventitia.

We will now examine an adult brain. On the adventitia of the veins fat is constantly found; I have found it without exception in every brain. At the least, we find in places single drops of fat, but nearly always evident fat-granule cells. The latter is present in great number, and give to the vessel a peculiar spotted appearance; not uncommonly the fat-granules form so numerous patches, that they surround the vessel for a larger or shorter piece with a perfect ring of fat (compare Fig. 2); this can be observed best in the smaller veins; but in the capillary vessels themselves fat-granules are by no means rare; their presence is a further proof that the capillaries possess their own lymph-sheath. I must here emphasise that these granule cells of the adventitia are situated always on the outside, accordingly they form a corresponding projection, as we see plainly in Fig. 2.

It may be interesting to note, that I have also found a considerable amount of fat in the cerebral veins of very emaciated subjects.

From the circumstances that these fat-granule cells are found in the adventitia of the cerebral veins in the new-born condition as well as in old age, and are seen in variable quantities after deaths from different causes (even after sudden and most violent death), I am justified in stating that the collection of fat on the adventitia of the cerebral veins, even when very abundant, is usually not to be considered a pathological occurrence; we see in these drops of fat and fat-granules (at least most of them) the remains of the period of embryonal development.

In case the fat-granules should be found in pathological changes of the central nervous system (as softening of the brain, &c.), they will be seen in great quantities on the vessels, vein and arteries. So, for example, we frequently observe in the spinal cord with degeneration of the posterior columns, even with the naked eye, the vessels which pass into the posterior commissure with their lateral branches presenting a thick white appearance. In such a case, where the surrounding nerve-tissue is diseased, the fat-granule cells on the adventitia are self-evident, as the expression of a pathological process, which attacks the vessels only secondarily.

In yet another way fat-globules can make their appearance on the adventitia (veins or arteries). It appears then on the inner side of the adventitia, which is clouded by a fine precipitate of very small drops of fat. This occurrence is rather rare, and appears to me to be the outcome of a morbid disturbance of nutrition.

We have spoken hitherto chiefly about the veins. When we prepare an *artery* from an adult brain, we find frequently, but not invariably, fat as in the adventitia of veins. On the other hand, the presence of pigment is constantly observed. This pigment, consisting of smaller or larger round globules, varying from light yellow to light brown, is found on the outer surface of the adventitia; frequently several of these granules are united with others to form aggregations (Fig. 1, *e*). In young children of two, three, or more years old, this pigment is very light yellow, or almost colourless, and shows all transitions up to the colourless fat-globules, mixed with which the pigment-granules are frequently found. This pigment hardly changes colour at all under the action of acids and alkalies; with osmic acid it takes a deeper colour and assumes rather a greyish tint; this reaction is more marked, the brighter the pigment has been before.

In my work on the cerebral vessels I have given a tabulated description of the condition of these vessels in ninety-one brains, and, with the exception of children under two years of age, I have never found pigment absent from the adventitia of the arteries. The relative quantity of pigment is such, that it can vary within certain limits, but it is perfectly independent of the causes of death, and cannot be referred to any morbid change in the individual in question.

I must particularise this point, for this pigment is always erroneously regarded as the effect of severe congestion of the brain, or even of actual hæmorrhage; it is confounded with the colouring matter of blood, which may also be found here, and I shall soon have much to say on this subject.

The question now arises, whence comes this pigment, which is found quite normally in the arterial adventitia of every brain, with the exception of young children?

I hold the opinion that this pigment arises from those fat-globules, which in early life are left behind on the adventitia of the cerebral vessels. Perhaps it represents a higher stage of oxidation of this fat, and arises by some change of tissue, through combustion of the fat.

Therefore we can speak of the following conditions:—

1. In very young subjects a very bright pigment is found, which resembles fat-granules in form and colour, and shows a stage of transition to the darker granules.

2. We often see on the arteries considerable collections, where fat and pigment are mixed together; also we nearly always find, in the same place, all the transitions from colourless fat-globules to yellow-brown granules.

3. The dark colour produced in the light-tinted pigment granules by the action of osmic acid, reminds us of the dark grey colour which fat receives from this reagent.

4. This pigment is also found on the veins; its proper situation, however, is on the adventitia of the arteries; in the arteries circulates blood rich in oxygen, here a more active change of tissue prevails, and on that account the change from fat into pigment has its seat here.

I must here add, to prevent misunderstanding, that I do not consider that the collections of fat found in the infantile brain must still be met with in the adult brain, as fat or pigment; a great part is perhaps absorbed in the mean time.

I will now take the opportunity of mentioning, yet another condition under which pigment may be found on the adventitia of the brain-vessels. True pigment cells are very frequently found on the delicate membranes of the brain (viz. on the medulla oblongata and base of brain). These cells are longish, for the most part with many thick processes, and with a nucleus which generally stands out bright in the midst of the finely granulated dark-brown pigment. This pigment is also seen on the larger vessels which pass into the base of the brain, they are merely introduced from the meninges. Not the least pathological significance can be attached to them.

Forms of pathological pigment on the cerebral vessels.

We have just seen that pigment is normally found on the brain-vessels under two forms.

There are deposits of pigment which can only be regarded as pathological, and particularly

1. *Blood-colouring matter, Hæmatoidin.*—This is seen as diffuse colouring matter of round granulated cells (fat-granule cells, or lymph-corpuscles), or in the form of larger or smaller red-brown bright granules; not uncommonly we find hæmatoidin crystals in the shape of rhombic columns. Such crystals or amorphous granules are frequently seen in the interior of the fat-granule cells. In isolated vessels we find the blood-colouring matter either applied to the adventitia or situated in its lymph-spaces; in cut sections we see that also the surrounding nerve tissue is nearly always richly pervaded with this pigment, namely with cells containing pigment. The presence of a large quantity of this pigment is always a sign that, in this place, an exsudation of blood has occurred.

The formation of hæmatoidin crystals as well as the diffuse yellow-coloured cells is easily recognisable; on the contrary, the granule-forms cannot be distinguished from normal pigment without further investigation. We possess a reaction which permits us, even after hardening in the chromate salts, to make out this difference without a doubt. The blood-colouring matter, under the influence of concentrated sulphuric acid, passes through a series of colours, viz. green, blue, and violet, and is finally discharged. Normal pigment hardly changes at all when thus treated.

2. *Malarial Pigment.*—In people who have suffered from attacks of intermittent fever the brain shows an almost dark grey colour, which is easily recognisable. This colour proceeds from a dark, nearly black colouring-matter; it represents granules which vary from the smallest dimensions to the size of a red blood-corpuscle.

Next we find these dark granules only in the blood, partly free in the plasma, partly in the interior of the blood-corpuscles. A considerable part of these pigment-granules can pass from the blood into the walls of the vessels, and fix themselves there; on the other hand, we do not find this intermittent pigment in the rest of the central nervous tissues.

Larger aggregations of this pigment can easily block one of the smallest vessels (pigment-emboli), whereby a rupture of the wall of the vessels ensues, and a small hæmorrhage occurs. This pigment is easily recognised by its dark colour, by its capability of resisting very different reagents, and above all by its presence in the blood itself.

We have therefore learnt to distinguish two kinds of normal and two kinds of pathological pigment on the vessels; I will presently bring forward an artificial pigment which has only had a short duration of existence. *Erlitzky* recommends as a hardening fluid a solution of bichromate of potash with sulphate of copper; this mixture found favour very quickly; and *Erlitzky* soon after described a spinal cord, which was hardened in his fluid, and pretends to have shown a very remarkable pigmentation (viz. in the vessels). I had originally a very doubtful opinion of this pigment,[1] but as a short time ago I saw a preparation of this kind, and have myself made observations with hardening in this fluid, I cannot have the least doubt about it that it is a precipitate, artificially caused by the hardening fluid. Since then, this appearance has been mentioned by others (W. v. Tschisch).

I take this opportunity of giving an example, how different artificial productions can be raised to the dignity of pathological formations, and I am justified in taking this example because *Erlitzky's* fluid appears to be generally used, and I would give a warning against further errors which might be produced by the action of this fluid.

Some histological changes of the brain-vessels, which occur under normal conditions.

After examining numerous cerebral vessels, I am convinced that there are in them a number of histological abnormalities, which, although they are considered to be pathological, are still met with in brains which, being in all other respects both anatomically and functionally healthy, must be considered quite normal. I will further add, that the changes presently to be described in the vessels need not produce any damaging influence, nor do they bring about in the living state any disturbance of function, or in the dead subject any anatomical lesion which can be connected with them.

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[1] Compare 'Centralblatt für Nervenheilk.' 1881, pp. 399–407.

This point is of great importance, from the fact that we should be forewarned of any judgment we may make from what we find by the microscope; we should not therefore, without further investigation, draw any conclusions from the presence of such vessels of any disturbance in the function of the brain.

1. *Deposit of calcareous matter on the vessels.*—Calcareous deposit (especially as carbonate of lime) can take place on the adventitia, as well as on the tunica muscularis.

In normal brains we do not often find chalk on the adventitia. If the calcareous deposit starts from the adventitia, it seldom remains limited to this membrane, generally the muscularis will be involved in the morbid change; but on the other hand the calcareous deposit can grow from the outside, as roundish or club-shaped chalk masses, which may attain a considerable size.

On the muscular coat I have frequently found chalk in perfectly healthy young subjects (viz. in girls 8 and 19 years old).

At first we see masses surrounding the muscular coat, with a peculiar slightly shining appearance; in further stages the whole muscular coat of an artery is changed through a longer portion of its course into a stiff finely granulated tube (Fig. 3). Even by macroscopical examination such a vessel can be seen by its white colour and needle-like rigidity. On moving it with a needle, the resistance is easily felt. Under the microscope we can recognise places where the calcareous cylinder is broken during preparation, and it shows cracks like broken glass. We are concerned here for the most part with carbonate of lime, and we can use in addition the reaction with sulphuric acid to make the diagnosis certain. By the reaction of this acid, bubbles of carbonic acid are formed, and fine needle-shaped crystals of calcic sulphate.

If the calcification is further advanced, we see the whole capillary network presenting a streaked appearance and changed into fine calcareous tubes. In the cerebellum, and especially in the granule layer of its cortex, such diffuse calcification can be repeatedly found, but it is not easy to say with certainty whether the adventitia or the muscular coat is the commencing point of the process.

We must suppose, *à priori*, that a calcification of the muscular coat will not only damage the nutrition of the brain, but, owing to the brittle condition of the vessels, will lead to further danger.

On the other hand I can state from experience, that with the exception of the higher stages of disease, such calcification, as has been mentioned, occurs very frequently under normal conditions; but hitherto, contrary to my expectations, I have not found any calcification of the muscular coat in cerebral hæmorrhage.

Calcification of the tunica media, if not very much advanced, has hardly any pathological significance.

2. *Fatty degeneration of the muscular coat.*—In preparations such arteries appear to the naked eye of an opaque yellow-white colour, without that rigidity which is found with calcification. Under the microscope we see the muscular layer filled with very small point-like fat-granules, and we can only here and there stain the muscle-nuclei slightly with carmine. Such a vessel shows under the microscope great similarity with a calcified one; except that the fatty muscular sheath is more yellow in colour, and never shows the fissures and cracks characteristic of the calcification; lastly, the sulphuric acid reaction is absent. This degeneration of the muscular coat we must also consider as a process which leads to serious functional impairment of a very important arterial covering.

In spite of this, I have met with this fatty change in healthy as often as in diseased brains; in old as well as in young subjects, and in those who have succumbed to acute as well as chronic diseases.

We cannot therefore attach great pathological meaning to this fatty change of the muscular coat.

3. *Connective tissue degeneration of the vessels.*—In arteries as well as in veins (the latter more frequently) an overgrowth of connective tissue proceeding from the middle coat (tunica media) of the artery takes place. The lumen of the tube is at first unaffected, while the circumference

of the vessels increases; thereby also the adventitia may remain intact. So we find pretty frequently spindle-shaped swellings of this nature on the little arteries or veins. It seems to me almost certain, that these enlargements of the vessels can usually be referred to the peculiar conditions of the blood-pressure which prevails at those places where a sudden change occurs in the calibre of the vessels; on this account they are not found so often at those places where the vessel divides dichotomously, but usually where the smallest vessels branch off immediately from relatively large trunks. A favourite place for finding the degenerated vessels is the highest layers of the cerebral cortex. *Neelsen* has shown,[1] that at this place these changed vessels are to be found in most brains, and I have also had the opportunity of convincing myself of this discovery.

Commonly we find, near the above-mentioned places, vessels in which the degeneration has made further progress. The connective-tissue overgrowth reaches at last the periphery of the vessel-lumen, the vessel is obliterated and continues its course as a cord of fibrous tissue. I have very frequently met with such vessels in perfectly healthy brains, even in children eight years old, but I must add, that this degeneration-process occurs much oftener and in a more marked degree in the vessels of old age (over sixty years), when once atrophy of the brain has commenced.

Although the blocking of the vessels caused by the fibrous overgrowth occurs gradually, it appears wonderful that a process by which individual regions—small though they be—of the brain are robbed of their nutritive arteries can be referred to a normal occurrence, and produce no known disturbance.

But on this point I should express myself with more caution. There are many obscure transitions in the domain of our cerebral life, some or other of which may perhaps be explained by local disturbances of nutrition, which comes about through such a blocking of vessels.

Deecke (loc. cit.) has confirmed the frequency of this change, for which he has used the expression "callous degeneration," and calls it a process situated "at the border of physiological and pathological conditions."

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[1] 'Archiv f. Heilk.' xvii., B.

4. *Dilatation of the adventitial lymph-sheath.*—Chiefly on arteries we occasionally see in places the adventitia, bulging like a blown-out bag, and separated some distance from the vessel. Such partial dilatations are seen in small as well as in large arteries. Fat-granule cells and pigment granules are often collected on such sacs.

Since such dilatations are often found in healthy brains, they must be considered as normal conditions.

If these bulgings of the lymph-sheath become very considerable and extending over numerous vessels, they must then be considered pathological.

More extended dilatations of the adventitia lead to the formation of cysts, which are visible to the naked eye. I have described the appearance of such cysts on the Pons Varolii, and they may be considered as cysts due to retention of fluid; the adventitial lymph-chamber is blocked by elements which have arrived in the lymph stream; whereby a stoppage of this lymph stream is produced, and hence the space becomes distended.

If the distension of the adventitia is diffused over many vessels and through a considerable tract of their course, the brain acquires, on cross-section visible to the naked eye, a peculiar sieve-like appearance studded with holes, a condition called "état criblé." This "état criblé" can arise through the enlargement of the perivascular lymph-spaces, as for instance through atrophic shrinking of the brain-substance; in this condition the adventitia, as well as the other coats of the vessels, remains unaltered.

The "état criblé" is chiefly confined to the white substance, whilst the cyst-formations as described above are only found in the grey matter.

Some important changes in the vessels which are decidedly pathological.

In the literature of this subject there are a very great many pathological changes of the brain-vessels which are certainly of very great interest.

I will, however, in the following pages only mention a few morbid appearances which seem to me of very great importance, purposely omitting the rarer points.

1. *The atheromatous fatty degeneration of the intima.*—This morbid process becomes evident by the presence of larger or smaller dark spots on the inside of the vessel (Fig. 5). Outside these spots we can readily recognise the healthy muscular layer; by pressure on the cover-glass it not unfrequently happens that these atheromatous masses are set free, float along further with the contents of the vessels, and finally become fixed on a narrow part of them.

We can also follow the formation of emboli in a small vessel from the point where it is liberated from the intima to where it blocks up a small branch.

By a higher magnifying power we see that these above-described deposits in the intima consist of a granulated amorphous mass, generally with very numerous fat-granules.

In nearly all cases of cerebral hæmorrhage I have been able to find this change in the intima, and this is particularly evident in the neighbourhood of the hæmorrhagic focus. I have not the least doubt that a great many of these cerebral hæmorrhages can be referred to this morbid change in the vessels, and especially that these atheromatous masses are torn from the intima to form emboli in the smaller branches.

We can also find this degeneration of the intima without any cerebral hæmorrhage, but this proves nothing against my view, as the atheromatous disease of the vessel must first be present, and then follows the embolism and hæmorrhage. I should therefore like to define the fatty-atheromatous degeneration in the intima as one of the most serious diseases of the smaller brain-vessels.

2. *The paralytic dilatation of the small brain-arteries.*—I have frequently found in brains, in chronic mental diseases, numerous arteries changed in the manner shown in Fig. 4. The muscular coat does not show a tube of even calibre, as it normally should, but it presents a succession of irregular, but not excessive, dilatations and constrictions; no other morbid changes can be recognised on the muscle-fibres,

even by using stronger magnifying powers. Most frequently I have found this condition of the arteries in cases of general paralysis. Later on, a chronic disturbance of the innervation of the brain-vessels may with great certainty be assumed to be caused by this disease, and I explain the above-described appearance as follows: that a partial paresis of the individual groups of the muscle-fibres in the tunica muscularis, or at least a diminution of the muscle-tonus, which after some time is produced, gives rise to these diffused enlargements of the vessels, which become permanent, and can be discovered after death. In the absence of any disease which can by other means be recognised, it seems to me that this explanation corresponds best to the conditions found.

This partial enlargement of the small arteries would therefore be considered as a sign of a chronic vaso-motor paralysis.

3. *Abnormal products contained in the lymph-space.*—Under normal conditions we find only a few formed elements in the adventitial lymph-space. We see single lymph-corpuscles, less frequently fat-granules, pigment granules, and rather large vesicular cells, which must be looked upon as metamorphosed lymph-corpuscles; exceptionally we chance to see a red blood-corpuscle. If the cover-glass be pressed upon, the above elements found between the muscular and adventitial coats float further on.

If we find numerous red blood-corpuscles in the adventitial lymph-space, their presence there is exceptionally due to diapedesis; more commonly it happens that a rupture has occurred in the inner coats of the vessel, and we have to do with a dissecting aneurism.

Very often we find an increase in the lymph-corpuscles in the adventitial lymph-space, their quantity can be so considerable that the muscular coat is so covered up with many layers of these elements as scarcely to be recognised. I consider that this increase of the lymph-corpuscles (improperly called "nuclei-growths") is to be referred to an emigration of white blood-corpuscles; we find it in different hyperæmic and inflammatory conditions of the brain. Also on the brain-vessels of general paralysis cases this increase is very frequent, as I have shown;[1] and just at this place the tendency to migration of the lymphoid elements is very remarkable.

Hitherto I have considered only the pathological increase of these elements, an occurrence which can also take place normally in the adventitial lymph-space; but we find there sometimes many kinds of pathological products, the presence of which shows a morbid condition. In purulent meningitis we can generally trace the pus-corpuscles along the vessel-sheath some distance into the interior of the brain; whether these corpuscles have had their origin on the spot, or have merely migrated out of the pia, it is frequently difficult or quite impossible to decide.

From a special pathological point of view, it seems to me that different kinds of neoplastic elements can be found in the adventitial lymph-space; I have seen there, cells of tubercular, sarcomatous, cancerous and syphilitic new growths. Such cells can be found easiest in the neighbourhood of a corresponding tumour. We may therefore conclude that the lymph-tracts of the brain afford a very convenient path for diffusing the various new growths, and the increase of a tumour often occurs through the intervention of this lymph-tract. In inflammatory processes, especially in the spinal cord, and also in lyssa, I have found round the arteries, in the adventitial and perivascular lymph-spaces, a peculiar regular-shaped substance without any recognisable structure, but colouring strongly with carmine. I believe that we must recognise in this substance the coagulation-product of a transudation proceeding from the blood.

I pass over a great number of changes of a different sort, which are to be found on the smaller brain-vessels.

The task which I have set myself to do has not consisted in giving a complete enumeration of all the diseases of the brain-vessels. I wish above all things to point out that much, which has passed current with us as morbid, is still within the confines of a normal state; with this I have thought it desirable to associate the discussion of some purely pathological

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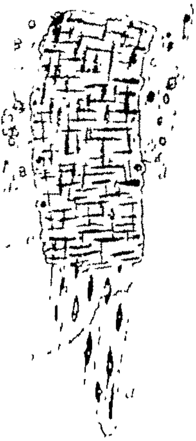
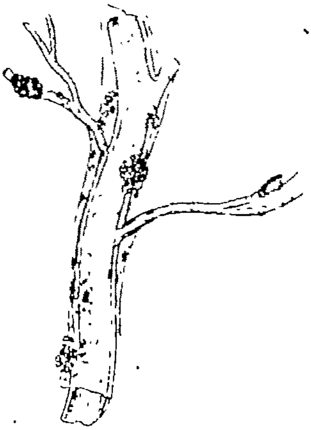
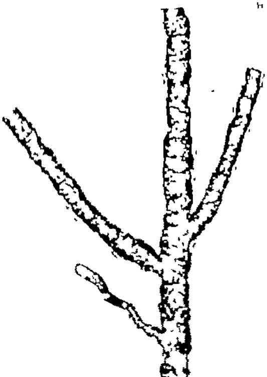
[1] 'Zur pathologischen Anatomie der Dementia Paralytica,' Virchow's 'Archiv,' 52 B.







conditions, which seemed to me of great importance from a scientific point of view.

EXPLANATION OF THE PLATE.

1. A small artery of the brain, torn in such a manner that the individual layers are visible.

a. Endothelial membrane.
b. Fenestrated membrane.
c. Muscular coat.
d. Adventitial lymph-sheath.
e. Little collections of pigment.

2. A normal artery, with fat-granule cells on the adventitia; these form in patches a perfect ring round the lateral twigs.

3. An artery in which the muscular coat is perfectly calcified, at the lower end of the chief branch the calcified tube becomes fractured.

4. An artery from the brain of a patient who died from general paralysis of the insane; we see, scattered about, little enlargements caused by the paralysis of the muscular coat.

5. An artery from the neighbourhood of an apoplectic focus; the intima has fatty-atheromatous degeneration, while the muscular and adventitial coats are intact.

(*Translated from original manuscript by* C. E. Beevor, M.D.)

ON THE EARLY OCCURRENCE OF ANKLE-CLONUS IN HEMIPLEGIA.

BY A. PITRES, M.D.,

Professor of Medicine to the Faculty of Bordeaux.

THE occurrence of ankle-clonus in hemiplegia of ancient date is a phenomenon both frequent and easy to demonstrate. Its semeiological and prognostic value has been shown by the researches of Professor Charcot, and we know that it usually coexists with late rigidity of the paralysed limbs and exaggeration of the knee-jerk. But accurate data concerning the exact time at which it may show itself after the apoplectic stroke are still wanting.

Professor Charcot has shown that in most cases the ankle-clonus precedes by a few days or weeks the appearance of secondary contractures (late rigidity). In a patient in his wards "ankle-clonus could be elicited eight days after the attack, and it was only a fortnight later—that is to say after a lapse of about three weeks—that late rigidity began to appear. In another case the foot-clonus became apparent only a month after the stroke, and rigidity supervened towards the end of the second month."[1]

Professor Charcot gives no further details on the point now under discussion. Professor Westphal is somewhat more explicit. According to him, ankle-clonus usually appears between the seventh and twenty-first day after the hemiplegic onslaught; and cannot be demonstrated immediately after the attack, nor during the next few days.[2]

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[1] Charcot, 'Leçons sur les Localisations dans les maladies du Cerveau.' Paris, 1876, p. 301.

[2] Westphal, 'Ueber einige Bewegungserscheinungen an gelähmten Gliedern.' 'Archiv für Psychiatrie und Nervenkrankheiten,' vol. v., 1875, p. 810.

From investigations to which I have submitted a certain number of hemiplegics admitted into my wards in the Hôpital Saint-André a few hours or a few days after the stroke, it results that the clonus may appear at a much more early date than Professor Westphal believes. In most cases, it is true, the phenomenon becomes observable only between the second and the sixth week; but it is by no means rare to witness it within the next few hours after the apoplectic seizure. Thus, in the two cases briefly recorded here, ankle-clonus was noticed eleven and fifteen hours respectively from the beginning.

CASE I.—*Cerebral lesion, followed with persisting hemiplegia. Ankle-clonus observed eleven hours after the accident.*

Mr. X., a law student, aged 22, was brought to the Hôpital Saint-André on the 4th of July, 1883, at 5 in the morning. He had received three-quarters of an hour previously, in a duel, a sword-thrust which penetrated into the right orbit. Immediately after being wounded he fell unconscious. Ten minutes afterwards he came to himself again, and from this moment a complete hemiplegia of the face and limbs on the left side was noticeable.

The regular examination of the patient was made at 3 P.M., eleven hours after the accident. Mr. X. is at this time in full possession of his faculties. He answers with precision to the questions addressed to him. The right superior eyelid is the seat of a marked ecchymotic tumefaction. The sword penetrated, outside the eyeball which was not perforated, at the level of the external cul-de-sac of the oculo-palpebral conjunctiva. It is uncertain how deep it went into the skull; the point was soiled with blood on a length of about five centimetres.

The left hemiplegia is total and complete. The mouth is deviated to the right, and is immovable on the left when the patient speaks. The limbs on the left side are inert and flaccid; they may be passively moved in every direction without showing any trace of rigidity. Voluntary movements are absolutely impossible. Sensation to pricking is diminished. The muscular sense is lost. When the eyes are closed, the patient cannot realise the position given to his limbs. Motion and sensation are normal on the right side.

The knee-jerk is much exaggerated on the left, slightly on the right. When the toes of the left foot are slightly raised a marked clonus ensues; the individual jerks are accentuated, and persist as long as the dorsal flexion is maintained. On the right side only two or three slight oscillations are obtained, which cease spontaneously though the foot be held firmly flexed.

From the 5th to the 7th of July the tendon phenomena remain the same as on the first day. No modification in the paralysis; no trace of rigidity.

During the next few days movement appears again in the left limbs, without any modification in the knee-jerks. The clonus when elicited on the left side remains the same; but it disappears on the right.

On the 16th, Mr. X. can stand and take a few tottering steps. He leaves the hospital.—We see him again on the 2nd of August and the 28th of October following. There is a rapid restoration of power in the left limbs. There is no appreciable rigidity, but the knee-jerk on that side is still much exaggerated, and the ankle-clonus as lively as on the first day.

On the 8th of July, 1884, we notice that the facial paralysis has almost entirely disappeared. Movements are possible with the left hand, but they are slow, clumsy, and lacking firmness. Muscular sense and tactile sensibility are profoundly altered; with the eyes closed Mr. X. does not realise the position given to his left hand, nor recognise a pencil or coin placed between his fingers. There is some permanent rigidity in all the articulations of the upper extremity.

The left leg is the seat of considerable rigidity. The dorsal flexion of the left foot causes a strong and persistent clonic trepidation, which is not observed on the right side. The left knee-jerk is much exaggerated; the right almost normal.

Dynamometric state of force	right arm,	53	kilogrammes.
	left arm,	36	" "
	right leg,	43	" "
	left leg	21	" "

CASE II.—*Right total and complete hemiplegia. Right ankle-clonus observed fifteen hours after the initial seizure.*

P. B., aged 66, had an apoplectic stroke with unconsciousness the 4th of June, 1883, at 2 A.M. He was taken to the hospital the same day at 5 P.M., fifteen hours therefore after the seizure. On entering the ward, patient seems still confused; he talks with difficulty, but can yet answer the questions addressed to him.

Right total and complete hemiplegia, without rigidity. The limbs are inert and flaccid. They may be moved in every direction without offering the least resistance; when lifted and left to themselves, they fall heavily on the bed. The right knee-jerk is much exaggerated, the left not so much. When the right foot is raised by the toes, a series of sudden and lively contractions is produced; but after six or eight oscillations this clonus ceases spontaneously, though the foot is kept dorsally flexed. No clonus on the left side.

June 5th.—Patient remains in the same state.

June 9th.—Clonus appears to be more easily elicited than on the first day. The oscillations are longer, and fourteen are counted before they cease spontaneously. No rigidity.

The patient returns to his home on the 10th. I see him a year afterwards: the hemiplegia has persisted, total and complete. The limbs on the right side are the seat of a marked late rigidity. Knee-jerk brisk and powerful on the right, normal on the left. Ankle-clonus very easily obtained on the right, and persisting as long as the foot is held up.

I have obtained evidence of ankle-clonus on several other patients admitted into the hospital from the second to the seventh day after the hemiplegic attack; but it is unnecessary to bring forward the observations in detail. The two cases just described suffice to show that the phenomenon is not always a late one, but may, on the contrary, appear a few hours after the seizure, at a period when secondary degenerations in the cord have not yet had time to be developed.

I shall add a few words concerning the prognostic value of the precocious ankle-clonus. I have hitherto seen only one case in which a patient presenting this sign recovered. It was that of a man, of 33 years of age and syphilitic, who, after having suffered from persistent headache, became hemiplegic on the night of the 18th to the 19th of December, 1882. He was admitted into the hospital thirty-five hours after the attack. He then had a total right hemiplegia with exaggerated knee-jerk on that side. Ankle-clonus was easily elicited in the right foot. An energetic antisyphilitic treatment was at once instituted. Ten days after, the clonus had disappeared, and the paralytical condition had already improved to a marked degree. Soon after, the patient left the hospital quite well.

With this exception all the other cases in which early ankle-clonus was observed have remained permanently and more or less completely hemiplegic, and with late rigidity of the affected leg.

It would thus seem, that the elicitation of ankle-clonus during the first few hours or days after the apoplectic attack is a symptom of evil omen, and an almost certain sign of an incurable paralysis.

CASES OF TUMOUR OF THE CORPUS CALLOSUM.

BY JOHN S. BRISTOWE, M.D., LL.D., F.R.S.

TUMOURS of the corpus callosum, so far as I know, are rare. But it has happened to me within the last three or four years to have under my care three cases of primary tumour of this organ, and another case in which it became involved in a growth springing from the floor of one of the lateral ventricles. Consequently it has seemed to me worth while to collect these cases; and to consider how far, if at all, the symptoms presented by them were distinctive.

The first case was that of a middle-aged man, who first complained of headache about fourteen weeks before his death. Shortly afterwards his right arm, and subsequently his leg, got weak; and his intelligence became impaired. The symptoms slowly increased upon him, the paralysis of the arm and leg became aggravated; indications of feebleness in the facial muscles appeared; he became apparently more and more stupid and extremely drowsy, and probably to a large extent on this account he ceased to speak, and allowed food to collect and lie in his mouth. Before death he passed into a state of complete coma. There were no fits, no actual sickness, and no affection of the ocular muscles. The chief phenomena, in fact, were headache, stupidity, and drowsiness, in association with paralysis of the arm and leg of one side, and doubtful paresis of the facial muscles of one or both sides—the symptoms being slowly progressive.

At the post-mortem examination a spongy, probably sarcomatous, growth was found to involve the corpus callosum, mainly in its anterior part, and to extend thence on either side into the centrum ovale, the left side being chiefly affected. There was no disease of the grey matter of the convolutions, or of the ganglia on the floor of the ventricles.

The second case also was that of a middle-aged man, whose illness came on while he was at sea, about ten weeks before it proved fatal. It is said that at first his speech became affected and his manner dull. When admitted, four weeks later, he had pretty complete right-sided hemiplegia, with involvement of the face and tongue, and well-marked aphasia. It was thought, indeed, that his symptoms were due either to hæmorrhage into the left side of the brain, or to plugging of the left middle cerebral artery. But the discovery of optic neuritis rendered it more probable that he was suffering from a cerebral tumour; and this view was confirmed by the slowly progressive aggravation of his symptoms. He gradually became absolutely speechless, and, though able to masticate and swallow, unable apparently to protrude his tongue, or to prevent the saliva dribbling from his mouth. He grew more and more dull and stupid, but not specially drowsy; and, though apparently not understanding anything that was said to him, stared intently and steadily at me, or possibly others about his bed. Before his death his pupils became unequal, rhythmical tremors attacked his arms, and his temperature rose.

This patient, like the other, complained of headache; but, also like the other, he never had a fit, did not suffer from sickness, and had no trace of paralysis of the ocular muscles. The chief symptoms were headache, right-sided paralysis, aphasia and dementia. Drowsiness was not specially noted in this case.

After death, a tumour was discovered, occupying and evidently originating in the anterior half of the corpus callosum, whence it extended into either centrum ovale. The left centrum was more extensively involved than the other, and on this side the grey matter of the second and third frontal convolutions and of the ascending parietal was involved. There was no disease elsewhere.

The third case occurred quite recently. This case was that of a middle-aged man. The history is not very clear; but he cannot have been ill altogether more than twelve weeks; and was probably ill for a much shorter period. When received into the hospital ten days before his death, he was partially paralysed in all his extremities, but mainly in the left arm and leg; there was doubtful paralysis on the left side of the face; he was very drowsy, and his speech was slow and indistinct, but he was sensible. During the progress of the case, the paralysis increased, mainly on the left side, and it was thought also that there was some weakness on the left side of the face; he became profoundly sleepy; he lost the power of speech; he manifested some difficulty in chewing and swallowing; and when awake he, like the last patient, stared intently but with a puzzled look at those about him.

This patient resembled the others in the absence of fits, sickness, and paralysis of the ocular muscles. His chief symptoms were, paralysis of the limbs, drowsiness, stupidity, loss of power of speech, and difficulty in getting rid of food placed in his mouth.

The post-mortem examination revealed the presence of a sarcomatous growth, infiltrating the anterior two-thirds of the fornix and corpus callosum, and extending from the latter into the white matter of both hemispheres, chiefly, however, into the right. Neither the grey matter of the surface nor the ganglia were involved.

The close resemblance, if not identity, between these cases as regards their morbid anatomy is obvious. In all of them the growth had clearly originated in the corpus callosum, and had thence extended almost symmetrically along the radiating fibres of this body into the white matter of the hemispheres. There is reason, indeed, to suspect them to be examples of a class of cases which, though attention has not been specially directed to them, are really not uncommon, and not unlikely therefore to be met with in practice.

There was also a considerable resemblance in the collective symptoms presented by them; and I am inclined to think these were sufficiently characteristic to suggest a correct diagnosis. At any rate my former experience enabled me, when the last case came under treatment, to make a happy guess as to its nature, and accurately to predict, as the corpse lay on the table, what would be found when the head was opened.

The chief characteristic features of the cases were:—1st, their ingravescent character, a character which they possessed in common with other cases of cerebral tumour; 2nd, the gradual coming on of hemiplegia, for the most part resembling in its distribution the paralytic symptoms usually attending hæmorrhage into one of the hemispheres, or softening due to embolism; 3rd, the association with the paralysis of one side of vague hemiplegic symptoms of the other; 4th, the supervention of stupidity, associated for the most part with extreme drowsiness, a puzzled enquiring look when awake, a difficulty of getting food down the throat, and cessation of speech—I say a difficulty of getting food out of the mouth rather than paralysis of the mouth and throat, and I say a cessation of speech rather than aphasia or loss of articulating power, because it seemed to me, in watching the cases, that these phenomena were due mainly to stupidity and irresistible tendency to sleep, and not definitely to paralysis or affection of the centres for speech; 5th, the absence of implication of the oculo-motor nerves, and of direct implication of other cerebral nerves; and lastly, death from coma. It is further noteworthy that some of the more striking symptoms of cerebral tumour were either absent or only slightly developed. Headache did not appear to be very severe in any case, and in one is not so much as mentioned; there was practically no sickness; optic neuritis was certainly absent, at any rate in one case, up to within a week or ten days of death; from first to last the patients were free from epileptic attacks, and never had anything that could be termed a fit.

So far as I know from reading, disease of the median portion of the corpus callosum is unattended with marked, certainly with characteristic, symptoms. And I am inclined to think that my cases are in accord with this statement. In all of them the tumour, taking its whole bulk, was very large; and must have taken much longer to grow than the clinical history gave as the duration of the disease. And in all of them the median part of the corpus callosum was evidently the starting-point of the disease, and this body must have been largely involved before the patient complained of definite cerebral symptoms. Indeed I take it that the symptoms the patients presented were due chiefly, if not altogether, to the extension of the disease into the hemispheres, and to the diffused pressure on important parts caused by the great collective bulk of the tumour, as evidenced by the extreme flattening of the surface of the hemispheres, and the singular absence of both subarachnoid and ventricular fluid. Assuming this explanation to be correct, the progress of a case of the disease I have been describing should be much as follows. First, the occurrence of headache, and other somewhat vague symptoms of progressive cerebral disease. Second, the gradual onset of more or less well-marked hemiplegia. Third, the appearance in a greater or less degree of similar symptoms on the opposite side of the body. Fourth, the coming on of dementia, with drowsiness, loss of speech, difficulty in swallowing, and want of control over the rectum and bladder. And these were, in fact, the chief symptoms which my cases presented, and the order of their sequence. Many other symptoms might of course become superadded in the progress of the case, owing either to accidental circumstances, or to the extension of disease into special parts of the brain. Thus, in the second of my cases, there was distinct aphasia, owing to direct involvement of Broca's convolution; and thus, although sickness and epilepsy were absent in the cases I have adduced, I see no reason why they should be absent from all cases of the kind.

The affections with which, as it seems to me, my cases would be most liable to be confounded are, hemiplegia due to hæmorrhage or embolism, disease of the medulla oblongata and its vicinity, and general paralysis of the insane.

I presume that if the case were observed from its very beginning, it would scarcely be possible to mistake one of embolism or hæmorrhage for one of progressive disease of the corpus callosum. And even if such a case came under observation late and without a history, its later progress would, I think, be sufficiently suggestive to the watchful physician. The second of my cases came to me with a very imperfect history; and, guided by the fact that he suffered from right hemiplegia with aphasia, I certainly assumed for the first few days that he was suffering from cerebral hæmorrhage or softening. But the steady downward progress of the case and the discovery of optic neuritis gave it a different aspect. The difficulty might be increased if the patient, suffering from a recent attack of embolic or hæmorrhagic paralysis, had had a previous attack of hemiplegia on the opposite side of the body.

Disease of the medulla oblongata and its neighbourhood has so far a resemblance to disease of the corpus callosum, that it is apt to be attended by so-called "cross-paralysis," and by difficulty in chewing, swallowing and speaking. But in this case the cross-paralysis is generally not the result of double hemiplegia, but the consequence of the association of hemiplegia with local paralyses due to direct involvement of one or more of the cerebral nerves. Or to put my meaning in another form: in disease beginning in the corpus callosum, if there be cross or double paralysis, it is owing to the co-existence of more or less complete right and left hemiplegia; whereas in disease of the medulla oblongata, the distinctive symptoms depend on the direct involvement of the nerves of that part, and we are apt to have paralysis not only of the muscles of articulation, mastication and deglutition, but also of the oculo-motor and other nerves.

At first thought, general paralysis of the insane might seem to have no relation to disease of the corpus callosum; and it must be admitted that the differences are, for the most part, much greater than the resemblances. Yet I am bound to confess that my third case seemed to me, when I first saw it, to be not unlike one of general paralysis; and it was only after watching the case for a day or two that I determined that it was not. The patient had incomplete paralysis of all his limbs; he passed his evacuations into the bed; he had (or said he had) difficulty in protruding his tongue; he spoke slowly and indistinctly; he seemed to masticate and swallow with difficulty; his language was flippant in quality; his face was extremely greasy; and he complained of nothing. Further, he had no optic neuritis, and no sickness. Now this combination of symptoms corresponds very fairly to what we meet with in certain cases of general paralysis, at certain stages of their progress. But, on the other hand, there was no inequality of pupils; there was no distinct tremor of the lips in speech; and moreover, his symptoms were those, if of any, of the latest stage of general paralysis, a stage which I suppose could not have been reached in the month or two during which alone the patient was ill, and certainly could not have been reached unless he had suffered from the epileptiform fits which generally attend the disease.

I have added the fourth case because, although it evidently does not form one of the same group as the other three, it has a close affinity to them, both in its morbid anatomy and in the symptoms which were observed during life. Indeed the symptoms, excepting that they were very much slower in their development, were almost identical with those of the other cases, and might well have justified the diagnosis of tumour starting from the corpus callosum.

The patient was a young woman, whose illness began with headache, giddiness and sickness, about fifteen or sixteen months before her death, which occurred about eight weeks after her admission into the hospital. To the above symptoms, which gradually increased upon her, failure of sight and difficulty in walking (with a tendency to fall backwards) were added in the course of time. On admission, she complained of headache, giddiness, and blindness; she could only walk with assistance, and had a manifest tendency to walk on her heels, and to fall over backwards. She was not sick. She had double optic neuritis. There was no definite paralysis, but the tendon reflexes in the legs were exaggerated. Her symptoms rapidly developed while she remained under observation; she became confined to bed; she passed her evacuations under her; her left arm and leg became manifestly paralysed and somewhat rigid; some weakness of the same side of the face was noticed; her speech became drawling and indistinct; extreme drowsiness came on, and with it a tendency to allow her food to remain indefinitely in the mouth. She died comatose.

The patient complained a good deal of headache, also occasionally had hallucinations, but otherwise was sensible; she was only sick once or twice while she was in the hospital, and she never had a fit. The oculo-motor nerves remained normal.

The post-mortem examination revealed the presence of a very large tumour, springing from the surface of the right corpus striatum and optic thalamus, and the neighbouring parts of the floor of the right lateral ventricle, distending this ventricle and displacing the fornix and septum lucidum (which were partly incorporated in it) to the left, and involving also the central and anterior part of the corpus callosum which lay over it.

The corpus callosum was much affected in this case, but I cannot attribute any of the patient's symptoms to this cause. The tumour of the ventricle, partly by the pressure which it exerted on the overlying right hemisphere, partly probably by its encroachment to a slight extent on the subjacent corpus striatum, furnishes ample explanation of the clinical phenomena of the case.

I am sorry to have to state, that the exact nature of the tumours in these cases was not (excepting in one instance) determined. I was present, however, at all the post-mortem examinations, and I believe that they were sarcomata.

CASE I.—*Tumour of Corpus Callosum extending mainly into left Centrum Ovale.—Right Hemiplegia.—Loss of memory and intelligence.—Drowsiness.—Coma.—Death.*

C. S., a bricklayer, aged 46, admitted Nov. 13th, 1880.

Has enjoyed good health up to ten weeks ago, when he began to suffer from severe frontal headache, and was compelled in consequence to give up work. He had no vomiting or other symptoms. He continued in this state up to three or four weeks before admission, when his right hand became weak, his memory began to fail, and his mind to be confused. It is said too that his sight became defective. His headache at the same time diminished. In the course of a week or so, the weakness of the right hand and arm had increased, the leg had become implicated, and his loss of intelligence had become more pronounced. About a fortnight since, he took to his bed, and at that time he fancied he was not in his own house. He has gradually been losing the power of walking and even of standing without assistance. He has been very drowsy in the daytime, but wakeful at night. He has felt nausea, but not been sick; and his appetite has been fair. He has retained power over his rectum and bladder.

He is a fairly well-nourished man; but his friends say that he has lost flesh. Cannot stand; but when supported in walking drags his right leg. He does not answer questions readily; will not do what he is told to do; and does not seem to understand all that is said to him; slow in thought and movement. Complains of pain in left frontal region. Face apparently a little smoother on right than on left side; but he opens and closes his eyes equally well; and the mouth is not drawn to left. Tongue protruded straight. No squinting; no double vision; says that he sees well; pupils equal; hearing good. Right arm and leg considerably weaker than left; no impairment of sensation. Patellar-tendon reflex increased in right leg. No affection of thoracic or abdominal organs. Tongue a little coated. Urine free from albumen. No loss of control over emunctories.

On the 18th he was very drowsy, lying with eyes half-closed, and refusing or unable to speak. The day before he was tested in reference to his knowledge of the names of things. He named objects fairly readily; and the only mistake he made was to call an inkstand a penholder. It was noticed that the left eye closed less quickly than the right. About this time he began to pass his evacuations into the bed.

He remained in much the same condition. And on the 28th it was noted that he was still drowsy, and could only be got to speak by repeating questions loudly two or three times; that he ate very slowly; and that he allowed food to remain in his mouth for five minutes or more without attempting to swallow it.

On December 3rd it was observed that the weakness of the right arm and leg had increased: and that the left eyelid did not close so perfectly as the right. He remained in the same drowsy state.

He gradually became weaker and more drowsy; and passed into a condition of complete coma, in which he died on the evening of December 8th. His temperature had been normal during the greater part of his stay in the hospital. But it rose to 100·6 on the evening of the 7th, and on the day of his death varied between 101·2 and 104·8. An hour before death it was 103·6.

Post-mortem examination.—All organs were free from disease except the brain. The membranes were healthy; and there was no excess of fluid on the surface, or in the ventricles. The vessels at the base also were healthy. A soft, spongy, vascular, reddish-brown tumour was found occupying the corpus callosum in the greater part of its extent, both from before backwards, and from side to side. It was largest anteriorly, where it occupied the anterior genu; and it extended thence on either side into the centrum ovale, chiefly however on the left side. On the right side it ceased opposite the extremity of the caudate nucleus: but on the left it extended to the expansion of the internal capsule. Neither the convolutions, nor the optic thalami, nor the corpora striata were involved. But the tumour adhered slightly to the upper surfaces of the anterior extremity of the left caudate nucleus.

CASE II.—*Tumour of anterior half of Corpus Callosum and each hemisphere, and on the left side involving the frontal convolutions.—Right Hemiplegia.—Aphasia.—Optic Neuritis.—Rhythmical Tremors.—Stupidity.—Death, with high temperature.*

Joseph McK., a sailor aged 41, was admitted under my care on the 19th of April, 1882.

Four weeks ago, while at sea, his speech became affected, and his manner dull. About the same time, or a little later, the right arm and leg got weak. He had no fit. The symptoms have increased upon him; and for two or three weeks he has kept his bed. He has not been emotional; and has retained power over his evacuations.

There is complete right hemiplegia. He cannot move either arm or leg; the mouth is drawn to the left; and the tongue is protruded towards the right. The leg is somewhat rigid, but the arm is limp. The tendon and superficial reflexes are normal on both sides; and there does not appear to be any impairment of sensation. No affection of ocular muscles; pupils contracted and equal; sight apparently good. No difficulty in deglutition. He can say a few words, and seems to understand fully what is said to him; but is evidently aphasic to an extreme degree. Does not repeat words to dictation. Hearing imperfect, especially on right side.

Tongue clean; no sickness; bowels open. Heart healthy. Urine free from albumen, sp. gr. 1025. Temperature normal.

At this time it was believed that the patient was suffering from an ordinary attack of hemiplegia due to hæmorrhage; but on the 24th his eyes were examined with the ophthalmoscope. It was then seen that the right optic disc was hazy and woolly, that some of the vessels had white lines along them, and that the veins were slightly dilated; but that the left eye was healthy.

27th.—He is in much the same condition, excepting that his loss of speech has become more marked. He now only says "no" in answer to every question. But, though there is doubtless some impairment of intelligence, he seems to understand all that is said to him, and tries to reply and do what he is told. He evidently recognises all objects that are shown him, and even their names when repeated to him. He answers "no," but at the same time shakes his head in negation, or nods affirmatively, as the case may be. He can copy letters imperfectly with his left hand, but cannot write. He does not recognise printed words or letters; at any rate he recognises them very imperfectly. He has some headache on the left side. The paralysis remains as before; but it is thought that there is also some impairment of sensation on the paralysed side.

A further examination with the ophthalmoscope was made by Mr. Nettleship. He confirms the description already given of the right disc; but he observes that the left disc is not absolutely clear; that at least three of the vessels bend abruptly just outside its margin; that there is slight neuritis.

From this time the change in the patient from day to day was slight; but on the whole he slowly got worse. He suffered more or less from headache, always referred to the left side. The paralysis of his limbs remained complete; but the arm after two or three weeks grew rigid; and the tendon and plantar reflexes in the right leg became exaggerated. No squint appeared, but the left pupil became at first smaller and subsequently larger than the right. A difficulty in protruding his tongue came on, and saliva at times dribbled from his mouth, but he could masticate and swallow. The slight hemianæsthesia continued. He lost the power of speech absolutely, and gradually became dull and stupid; and, though looking at and watching intently any one who addressed him, apparently made no effort to respond. Occasionally he seemed fretful; but on the whole he was utterly apathetic. The pain in his head was relieved once or twice by the application of a few leeches to the temples.

On the 20th of May the following note was taken:—The patient lies in an apathetic condition, taking very little notice of anything, and seeming not to understand when spoken to. His chief employment is gnawing a flannel roller or his handkerchief. The paralysis remains complete; and the arm, but not the leg, is rigid. He seems unable to put out his tongue, and is wholly speechless. But he can swallow, and has a good appetite. The pupils are equally dilated, but from the effects of atropine. He apparently has pain on the right side of the head. His evacuations are passed unconsciously. For the first time his temperature rose to-day to 100·8.

From this day he gradually sank. He got more and more dull and stupid. Rhythmical tremors came on in his arms, but only when they were moved voluntarily or by force. The pupils became unequal, the right being the smaller. Profuse perspirations broke out, and the temperature rose. On the 26th it reached 102; on the 27th, 103; on the 28th, 105·4; and just before his death (which occurred a little after 8 P.M. on the 29th), 107·4.

Post-mortem examination.—Dura mater normal. There was no excess of fluid either in the subarachnoid tissue or in the ventricles, and no trace of meningitis. The surface of the left cerebral hemisphere was perhaps a little more prominent than that of its fellow. On making careful transverse and longitudinal sections of the brain, a large infiltrating ill-defined growth was found, involving the anterior half of the corpus callosum; and extending thence, on the left side into the substance of the hemisphere, so as to occupy nearly the whole of the anterior half of its white matter, together with the grey matter of the second and third frontal convolutions and the ascending frontal; and on the right side so as to implicate a considerable portion of the right centrum ovale in the corresponding situation, but not reaching the grey matter. The tumour was above the level of the corpora striata and optic thalami, and did not affect them, or the internal capsules. The new growth was greyish in tint, and looked soft. But it was really tough, and cut crisply. The portion occupying the left hemisphere was spongy; that on the right side was more homogeneous in texture, but presented a series of concentric markings.

All the other organs were healthy.

CASE III.—*Tumour of anterior three-fourths of Corpus Callosum, extending into the white matter of the hemispheres.—Imperfect paralysis, mainly of the left side; difficulty of speech; great drowsiness and stupidity.—Death.*

J. H. P., an accountant, 51 years of age, was admitted under my care on the 21st of July, 1884.

He had been a strong and healthy man until ten weeks previously, when he was attacked with quinsy. He was ill with this complaint for a fortnight or more, and remained feeble for a few weeks longer. But he finally got quite well; and is said to have remained well until ten days before admission. At that time it was supposed that he was overcome by the heat of the weather; at any rate he became weak, and his weakness has increased from that time. He was sent to the hospital with the statement, that he was suffering from diphtheritic paralysis.

A well-nourished man. He was very drowsy, but when fully awake appeared to be quite sensible, and, though speaking slowly, made pertinent, indeed somewhat flippant, answers to questions. But his articulation was indistinct. He did not fully open his mouth or put out his tongue when told to do so, and said that he could not. The right side of the face was thought to be a little less mobile than the left. There was loss of power in both upper extremities; and the left was flexed and somewhat rigid. The legs also were weak. The right he seemed unable to move, the left he moved slightly. The tendon reflexes were present and normal; as also were the cutaneous reflexes. There was no loss of sensation anywhere. There was no nystagmus; no squint. The pupils were of normal size, equal and acted to light. His urine was passed into the bed.

No affection of heart or lungs. Pulse 80. Abdominal organs all healthy.

I saw him the next day, and his condition accorded in the main with the above description. But his drowsiness had increased; and it was very difficult to wake him or to keep him awake. He seemed to have a little difficulty in swallowing, and in getting the food out of his mouth. This, however, might have been due to his sleepiness. He answered questions only when fully roused, and then sensibly, though slowly and somewhat indistinctly. There did not appear to be any actual aphasia. He said that he had little or no pain. The eyes were quite normal as to pupils and movement; and ophthalmoscopic examination revealed nothing amiss. The evacuations were passed unconsciously; and the urine, which had a specific gravity of 1032, was acid, and free from albumen and sugar.

He continued in much the same state for the next two or three days. On the 26th I made the following note:—"Patient still passes everything into the bed. He is much less drowsy than when I last saw him. Hears, but does not seem to understand what is said to him. When spoken to loudly, makes some attempt to do what he is told or to answer, but his speech is mumbling and unintelligible. He keeps his eyes intently fixed on my face, and follows me with them when I move. He has a little difficulty in chewing and swallowing, and occasionally chokes. There is no paralysis of ocular muscles, or affection of the pupils. The left side of the face is perhaps a little weaker than the right. Moves right arm more than left, but both are weak and somewhat rigid. Legs as before. Sensation perfect. Face very greasy."

Henceforth his symptoms underwent scarcely any change. He became perhaps more drowsy and difficult to wake, and difficult to feed on account of his drowsiness. There was no obvious increase of paralysis; but certainly no improvement in this respect; and his evacuations were still discharged into the bed. He occasionally tried to speak; but the sounds he uttered were unintelligible. On the 28th his pulse was 112, but his breathing normal. On the 29th his urine, which was still free from sugar and albumen, was strongly alkaline, and its specific gravity 1035. On the evening of the 30th he had an attack of dyspnœa, attended with rattling in the throat; and the respirations rose to 54 in the minute. The dyspnœa subsided in the course of a few hours. During the whole of the 31st he was unconscious. It was noticed that both arms, but chiefly the left, were rigid. At six in the evening he began to breathe stertorously, and he died three hours later.

It is noticeable that the patient never complained of, or seemed to suffer from, headache; that there was never any sickness; and that he had never had anything of the nature of a fit. His temperature, during the first three days, varied between 98·4 and 99·8. During the next seven days, it rose every evening to between 100 and 100·6. Shortly before death it attained 102·2.

Autopsy.—On removing the dura mater the convolutions were found universally flattened, the surface of the brain dry, and the pia mater and veins everywhere remarkably congested. But there was no appearance of inflammatory effusion and there were no adhesions. No fluid was found at the base of the skull; and none escaped from the lateral ventricles as the brain was removed. The arteries were all healthy. On separating the hemispheres, the corpus callosum was seen to be much arched from before backwards; and the surface of its anterior two-thirds, as far outwards on either side as it could be traced, was yellowish and irregularly modulated. On further examination, it was found that the anterior three-fourths of the corpus callosum, and the corresponding portion of the fornix (including the anterior genu of the former, and the anterior pillars of the latter) were the seat of a growth, which had caused the affected part of the corpus callosum to be at least as thick again as natural, and the fornix to measure in some places about half an inch in thickness. The general form of these bodies was, however, retained. The diseased fornix was grey, translucent, soft and vascular, and had the appearance of being the seat of growing sarcoma. The corpus callosum presented patches of the same kind of material, but was mainly the seat of a toughish opaque buff-coloured reticulum with coarse irregular meshes, occupied in part by the growth above described, in part by a gelatinous kind of material, or a serous fluid. The diseased textures extended from the corpus callosum on either side into the anterior half of the centrum ovale majus, which was involved in almost its whole extent above the level of the corpus striatum. The disease was somewhat more extensive and more advanced on the right than on the left side; and the affected tissues had exactly the same characters as those presented by the central part of the corpus callosum. There was no very definite limit between the tumours and the brain-tissue around; which latter was soft and much congested. Microscopic examination showed that the tumour was a sarcoma, undergoing in places degenerative changes. The grey matter of the convolutions was everywhere healthy; the ganglia at the base were also altogether healthy; and the lateral ventricles contained only a minute quantity of fluid. There was no disease elsewhere in the brain; or in the body.

CASE IV.—*Tumour springing from floor of right cerebral ventricle, and involving the septum lucidum, fornix, and corpus callosum.—Headache, optic neuritis, blindness, left hemiplegia, drowsiness, coma, death.*

Florence M., an unmarried woman 25 years of age, was admitted into St. Thomas's under my care on the 13th of June, 1883.

She is said to have been a delicate woman, but to have suffered from no serious illness until fourteen months ago, when she had a sharp attack of small-pox. She dates her present illness from that time. The first symptoms were pain at the top of the head and across the eyes, giddiness, sickness, mainly in the morning, and weakness in the legs. These symptoms gradually became more severe; and latterly her eyesight has been failing. For some time past, when standing, she has had a tendency to fall backwards. She has been quite sensible all along, and has had no fits. No history or sign of syphilis.

She is a florid, healthy-looking woman, perfectly sensible, and ready of speech. She complains of vertex headache, of blindness, and of difficulty in walking. The headache varies, but is never severe. The eyes are very prominent, but otherwise look natural. Their movements are consensual, and normal, excepting that the corneæ are never turned completely into the outer or inner canthi. The pupils are equal, but act neither to light nor to accommodation. Sight is almost gone; the patient can only distinguish light from darkness. There is double optic neuritis; the discs are much swollen, but little congested, the vessels are tortuous and slightly obscured at the discs. There is no paralysis of either side of face or tongue, and mastication and deglutition are unaffected. Sensation is perfect in head and face. Hearing and taste are normal. Says she has lost smell, but she recognises ammonia and acetic acid. There is no paralysis, rigidity, tremor or wasting of arms or legs. But the legs present exaggeration of plantar and tendon-reflexes, with clonus at ankles and knees. There is no apparent impairment of sensation. But she says she feels as if walking on wool. She can stand and walk, though with difficulty. The gait is a little peculiar. There is no ataxia, but she walks very much on her heels, and has a constant tendency to fall backwards with an inclination to the right. There is no affection of bladder or rectum, and the catamenia are regular. No bed-sores. Thoracic and abdominal organs apparently all healthy. Urine free from albumen.

The patient rapidly got worse after admission. In three or four days it was found that she could not stand without being supported on both sides. On the 18th it was noticed that she was excited; and on that day and for several days subsequently she had optical hallucinations, addressed imaginary persons, and said she saw soldiers all dressed in blue. This condition disappeared after a few days, and apparently did not recur. Then and thenceforth she was emotional, sometimes low-spirited, but more frequently jovial. On the 23rd and 24th, she passed her evacuations unconsciously, and for the most part they escaped without control during the remainder of her life.

Early in July she began to suffer from drowsiness. About the 8th it was observed that the mouth was drawn slightly to the right when she laughed or spoke; that the right eye closed a little more than the other when she smiled; and that her speech was slow and drawling.

On the 15th, it was remarked that the right arm seemed to be weaker than it had been: and that the left was occasionally rigid.

Subsequent observation confirmed the fact of slight left facial palsy; and that of comparative weakness with rigidity of the left arm and leg, the rigidity being occasional. Once or twice the tongue was protruded towards the left.

Her headache continued, and on two or three occasions she was sick. From the time of its commencement, her drowsiness, with occasional intermissions, continued, and increased upon her. And for the last week or two of her life it was so constant and so profound that there was the greatest difficulty in rousing her even for a moment; and it became a long and weary work to give her nourishment, inasmuch as she went to sleep with the food in her mouth, and if left, would allow the food to remain there for half an hour or more at a time. She generally lay with the tongue lolling out of her mouth.

Finally her drowsiness passed into coma, and in this condition she died early in the morning of the 5th of August.

She never had a fit. Her temperature throughout her illness was generally normal. Occasionally, however, it rose to 100, and shortly before death reached 100·8.

Autopsy.—A well-nourished young woman, with very crooked legs, the consequence of rickets.

On opening the head, the convolutions were found to be flattened, the surface generally dry, and the pia-mater uniformly congested. On removing the brain, there was no accumulation of fluid at the base, and no visible sign of inflammation; but it was noticed that the space between the crura cerebri was widened, and that the optic tracts were stretched. The infundibulum, however, was not prominent, and no fluid oozed from this part. On slicing away the upper part of the hemispheres, the corpus callosum was found to be unusually arched from before backwards, and also from side to side, as though raised by some subjacent accumulation, situated mainly towards its anterior part. The dissection was now conducted with care, and the following are the results which were arrived at:—A very soft, pinkish, vascular growth, much lobulated on the surface, measuring about 4½ inches from before backwards, 3½ from side to side, and from 1 to 2½ in thickness, sprung mushroom-like from the surface of the right corpus striatum and anterior part of the right optic thalamus, and from the floor of the anterior cornu and of the ventricle external to the ganglia. The subjacent brain-substance was scarcely, if at all, encroached on by it. The growth occupied the whole of the distended right ventricle, excepting its descending and posterior cornua; and, pushing the septum lucidum over to the left side, stretching it, and incorporating it to a large extent, formed a lobulated protrusion into the left ventricle. The fornix also was largely displaced to the left and stretched over the tumour; and the greater part of it, like the septum, was incorporated in it and lost. The corpus callosum, in its central area, was stretched over the tumour, adherent to it, and partly destroyed by it. Although the tumour had obviously originated in the right ventricle, about a fourth of the bulk was to the left of the mesial line. The third ventricle was a little displaced, but normal in all other respects. There was very little fluid in the cavities. The brain, with the exceptions above noted, was quite healthy. The tumour, judging from hasty examination, was a sarcoma.

Abdominal and thoracic viscera all healthy.

MUSCULAR ATROPHY, AFTER MEASLES, IN THREE MEMBERS OF A FAMILY.

BY J. A. ORMEROD, M.D.

The two children whose cases form the principal part of this paper were under observation, as out-patients at the National Hospital for the Paralysed and Epileptic, from February to September 1883. In August 1884, I heard from their mother that they were still in the same condition. They had been, at an earlier stage, under the care of Dr. Barlow, who had noted the paralysis as peculiar, and who has kindly seen them for me again.

Case I.—Annie W., æt. 17, was brought by her mother to me on January 30, 1883. Her legs were crippled, though she was able to walk; both hands were partially paralysed. The history given was this: she had measles between 6 and 7 years of age; about a month after she recovered from them, one foot (the right ?) began to "turn," the toes, according to the mother, pointing downwards and inwards. Tenotomy was performed at the Orthopædic Hospital. After this the other foot began to turn in the same way. About seven years ago (*i.e.* three or four years after the original attack of measles) tenotomy was again performed at University College. This affection of the legs did not come on suddenly, but took about a month to develop.

More recently the hands have become weak. This began five years ago. The mother first noticed that the girl used her hands awkwardly and stuck her knuckles out. The hands have got gradually worse, especially the thumbs, during the last two months.

She is subject to headache; also to shortness of breath and wheezing at the chest. Her menses are regular and natural, but preceded by much pain across the back and lower abdomen. With these exceptions her general health is perfectly good.

Her condition while under observation was as follows. Her legs were much wasted below the knees; the muscles of the thighs were not so well nourished as they should be. In cold weather there was much disturbance of the cutaneous blood-supply in the legs; sometimes the calves were blue and cold, with bright red patches on them like chilblains going to break; sometimes, in addition to the coldness and congestion, the whole leg below the knee was so swollen as quite to hide the wasting of the muscles. The tendon-reactions were present at both knees. The induction current produced no action when applied to the muscles below the knees, to the peroneal nerves—to the vasti—to the muscles at the back of the thighs (the last doubtful). The constant current also produced no action when applied to the peroneal nerves, or (so far as could be made out) to the muscles below the knees.

As to the condition of the hands and arms, for which she principally sought advice—there was well-marked wasting of the balls of both thumbs and of the intrinsic muscles of the hands generally; and wasting, though less apparent, of the muscles on the forearms, both extensor and flexor aspect. The fingers were in a position of semi-flexion at their two distal joints; their metacarpo-phalangeal joints being almost straight. All voluntary movements at the elbow- and shoulder-joints were perfectly retained; but extension of the wrist and fingers was possible, on the right side, only to a very slight degree; on the left side, not at all. Flexion of the wrists and fingers was possible, but to a slight degree only, on both sides. The left hand was said to be the worst.

Electrical examination, on two or three occasions, (it should be mentioned that she could not bear very strong currents) gave the following results:

(*a*) The induction current produced no action in the muscles of the thumb-ball, or intrinsic muscles of the hands, nor in the muscles of the forearms; on the other hand, the muscles of the upper arms, and the deltoids, rhomboids, &c., acted normally.

(β) Reaction to constant current.

In muscles of thumb-balls present but diminished[1] [on L. side AOZ = KSZ: on R. side ASZ = KSZ]: later, no reaction could be obtained.

In extensor muscles of forearms: on L. side, absent: on R. side diminished, without qualitative change: later, no reaction on either side.

In flexors: on L. side absent; on R. side, diminished without qualitative change. But the flexor longus pollicis and pronator radii teres seemed to act normally.

In ulnar nerve at elbow, and median nerve at wrist, no reaction.

To summarize; the affected muscles showed absence of farado-contractility, and absence or diminution of galvano-contractility, with qualitative change in one instance only.

From Jan. 30th to May she took codliver-oil and syrup of phosphate of iron; from May to September, hypophosphite of soda. Meanwhile she had the constant current to the upper limbs. Complaint was made in July of incontinence of urine, not at night, but while she was walking about. This had been noticed once before; but it did not appear to trouble her much. In the condition of her limbs there was no change; and her attendances became so irregular that she had to be discharged.

Case II.—Alfred W., æt. 9, brother of last patient, was first brought on Feb. 20, 1884, the history being that four years ago, after measles, weakness of both legs set in. This came on gradually, in about three months' time. But during the last few weeks, his mother had thought that the hands were getting weak.

The muscles of the lower limbs were much wasted below the knees, above they were apparently healthy; the patellar tendon reactions were exaggerated on both sides. The right thumb-ball was flattened as compared with the left; the induction current acted on *neither* thumb-ball. He appeared slightly feverish, and after this visit was laid up at home with bronchitis, so that I did not see him again till April. He had then got thinner, and his mother thought that his hands were worse.

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[1] *I.e.* required a considerably stronger current than the corresponding muscles in my own person. The estimation was made with a galvanometer, to avoid fallacies due to inaction of cells and variations of cutaneous resistance.

The following notes were taken between April and September.

There is wasting of both lower limbs, worst on the right side; but only below the knees. There is no vaso-motor disturbance, as in his sister's case. He wears orthopædic boots; when he walks without them, the right foot turns in. He can flex and extend the knee-joints freely; the patellar tendon-reactions are lively on both sides. As to the upper limbs, there is now evident wasting of the muscles of *both* thumb-balls, but still most marked on the right side. He cannot move the right thumb so freely as the left. The fingers tend to assume a position of flexion at their two distal joints, and extension at their metacarpo-phalangeal joints. This is particularly noticeable while he is using the hands. It is exactly the pose[1] which the mother noticed in an early stage of his sister's disease. He can, however, flex and extend the fingers at will. The forearms and upper arms are unaffected; the tendon reactions at wrists and elbows are lively.

Systematic electrical examination was impossible, on account of the child's nervousness, but the following facts were made out.

(*a*) A fairly strong induction current (which acted readily on my own thumb-muscles) had no action on his thumb-muscles, nor on the first dorsal interossei (the latter doubtful), but acted on the muscles of the forearms, both extensor and flexor aspect. It did not act on the muscles below the knees, either anterior or exterior group, nor yet on the vasti, though these latter did not appear wasted.

(β) A moderate constant current acted on the muscles of the right[2] forearm, extensor aspect, KSZ > ASZ; but had no action on the thumb balls.

The constant current did not act on the muscles of the legs, but acted on some at least of the thigh muscles.

The electrical conditions then, so far as they could be made out, were the same as in his sister's case; viz. absence or much diminution of contractility, in the affected muscles, to *both* currents.

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[1] Due to paresis of the interossei.

[2] The left forearm, and the other muscles of the right, were not examined, but may perhaps, from their normal appearance and faradic reactions, be presumed normal also.

He was treated with faradisation to his hands and forearms, and with the same medicines as his sister. He did not improve; indeed the peculiar pose of his hands became, if anything, more marked.

In neither of these cases was any complaint made of sensory disturbance. There were no fibrillary twitchings, and no rigidity. The affected muscles were, as I have said, wasted or wasting; and I did not notice enlargement of any other muscles; though I admit that I did not specially look for it. Being told by the mother that the father had been paralysed in one leg from infancy, I visited him at his cottage, and made the following notes.

CASE III.—William W., æt. 56, cab-cleaner, healthy in appearance, though grizzled and looking older than his years. Ever since he can remember, his right leg has been in its present condition. He was told by an elder sister[1] that he had measles very badly when 12 months old, and that the right foot began to turn when he was about three years old. His family seem to have connected the affection of the leg with the measles.

There is a decided difference in girth between the two lower limbs, especially below the knee, as follows:

Largest girth of left calf, 12·5 inches .. of right, 9·5 inches,
„ „ „ thigh, 16 „ .. „ „ 15 „

In length, no difference between the two. Patellar tendon reactions well marked on both sides, but the left greater than the right. The muscles below the right knee are extremely feeble; the foot hangs quite limp at the ankle, he cannot point the toes at all; he says he always walks on the heel; and he can only evert the foot slightly.

He cannot extend the right knee-joint with the same facility as the left.

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[1] This was told him before the birth of his two children, now paralysed. I mention this to show, that the sequence of events in his case was not made to suit theirs.

The left leg is perfectly natural, the arms and hands are natural, and he is positive that he never had anything the matter with them. I had no opportunity of making an electrical examination.

Evidently his condition may have been due to an old attack of infantile paralysis, but I do not feel certain that it was so. The history, such as it was, pointed to a gradual onset; and the bones had grown to the same length as those of the other limb. In former years a doctor has taken much interest in his condition, had him photographed, &c.

Of five other children (besides my two patients), one was smothered in bed, four are alive and healthy. Two of these are grown up. All had measles at one time or another. The mother was one of a remarkably large family of children, and she knows of no nervous complaint among them. The father (Case III.) was one of four, all healthy; of these a sister committed suicide at 15, but he is confident she was provoked to it, and was not insane. His father, mother, and paternal grandfather, were all healthy.

The points that deserve notice are

(1.) The connection with measles.

(2.) The youth of the patients.

(3.) The fact that brother, sister, and father were paralysed.

(4.) The possible nature of the disease.

The measles were not apparently severe in the children's case. No doctor was called in; but from the description the mother gave me of the coryza and the rash, and from the fact that measles was known to be in the neighbourhood, I have little doubt that this disease was, as she said, measles. The two children who became paralysed did not have them at the same time; one of the others had them at the same time as Alfred, but did not become paralysed; neither did any of the neighbours' children, so far as was known to the mother. There was therefore nothing in the nature of the epidemic specially tending to cause paralysis. On the other hand, considering the uncommon nature of the paralysis, it seems unlikely that the connection with measles should have been in the case of both children (and in that of their father?) a mere chance sequence. The exciting cause was probably the measles; the predisposing cause, a family proclivity to this form of paralysis.

This brings us to the other points, which it will be convenient to consider together. What was the nature of this paralysis? The three clinical types which suggest themselves are: (1) acute anterior poliomyelitis or ordinary infantile paralysis; (2) chronic[1] anterior poliomyelitis or paralysie spinale antérieure subaigue (Duchenne); (3) progressive muscular atrophy.[2] We may dismiss the first of these, with which the onset and course of the disease entirely disagree. As to the second, chronic anterior poliomyelitis, the youth of the two patients might have been held, till recently, to justify us in dismissing this also. But cases recently published[3] by Professor Erb and Dr. Hughes Bennett show that chronic anterior poliomyelitis does occur in children, however rarely. Indeed, between Dr. Bennett's first case and my own there are some close similarities. Dr. Bennett's patient suffered from whooping-cough and *measles*; a month afterwards, slight weakness and unsteadiness of the gait was noticed; still later, awkwardness of the hands; there was cyanosis of the legs, and diminished response both to faradism and galvanism. There remains, however, this difference, that in my patients there has been no attempt at recovery, though the disease is now of five and ten years' standing; whereas both Erb and Bennett emphasise the fact that their patients recovered, as being confirmatory of the diagnosis.

Moreover if it is rare for chronic anterior poliomyelitis to occur in children, it is, so far as I am aware, unknown that it should run in families. This is not the case with progressive muscular atrophy; which, as is well known, frequently attacks many members of a family. These also are just the instances in which the disease begins in early life. For recent information on the subject of "hereditary" muscular atrophy, I may refer the reader to an article by Zimmerlin, recently abstracted by Dr. Beevor.[4] As regards the progressive muscular atrophy of youth, Erb recognises this as a distinct type and assigns to it the following characteristics.[1] It begins insidiously in childhood or youth, usually in the shoulder or arm muscles, sometimes in the legs or back. The pectorals trapezii latissimi rhomboids and long muscles of the back are at a certain stage almost always involved; biceps, brachialis, and supinator longus very frequently; and the triceps later. Among other muscles, the deltoids and *small muscles of the hands are always spared*, and the muscles of the forearms for a long time. In the lower extremity, the glutæi, quadriceps, peroneals and tibialis anticus are usually attacked. The districts of the bulbar and cerebral nerves are not invaded.[2] While some muscles are soft and flabby, others may remain firm. There are no fibrillary twitchings. The irritability of the muscle to mechanical stimuli disappears. The patellar tendon reflexes are preserved. Distinct reaction of degeneration has never been demonstrated to exist.[3] The course of the disease is very chronic. This form of disease, Erb suggests, when it attacks several members of one family, constitutes the "hereditary" or "family" form of progressive muscular atrophy; and when it is complicated with formation of fat in lieu of the atrophied muscle, constitutes pseudo-hypertrophic muscular paralysis, which is also, as is well known, a "family" disease. He thinks that the disease begins in the muscles themselves, not in the cord or nerves.[4]

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[1] Better perhaps called subacute, to avoid confusion with (3).

[2] This last probably comprehends several forms.

[3] 'BRAIN,' Nos. 21 & 23, "On Chronic Atrophic Spinal Paralysis in Children."

[4] Zimmerlin, 'Zeitschrift für Klin. Med.,' Band I., Heft I. 'BRAIN,' No. 26, p. 285.

Now as regards the present cases, the age of the patients, the family predisposition, the absence of distinct reaction of degeneration,[5] the preservation of the tendon reactions—all suggest that they should be classed as instances of this juvenile form of progressive muscular atrophy. But the order of invasion, at least as regards the upper limbs (first muscles of hands, then of forearms), certainly does not agree with this view.

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[1] The original article will be found in 'Deutsch. Archiv für Klin. Med.,' Band 34, s. 467. I quote from an abstract by Strümpell, 'Neurolog. Centralblatt,' 1884, p. 200.

[2] Duchenne states that progressive muscular atrophy in children begins in the face. E. Remak ('Neurolog. Centralblatt,' 1884, p. 337) gives a case of Erb's juvenile type, with complete facial diplegia.

[3] There is a simple diminution of contractility to both currents ('Neurolog. Centralblatt,' 1883, p. 458).

[4] Recent pathological observations by Landouzy and Dejerine confirm this. See 'Lancet,' Jan. 26, 1884.

[5] Qualitative change was found once only the galvano-contractility was diminished or absent; see above.

I am compelled therefore to leave the exact nature of the disease undecided; perhaps the further course of the cases may decide it; but as it seems unlikely that I shall have further opportunities of investigating them, I venture to record them as they at present stand.

LESIONS OF THE NERVOUS SYSTEM ETIOLOGICALLY RELATED TO CUTANEOUS DISEASE.

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THE following observations are founded chiefly upon the following works:—'Die Neuropathischen Dermatonosen,' by Dr. Ernst Schwimmer, of Buda-Pest; 'Recherches Cliniques et Anatomo-pathologiques sur les Affections Cutanées d'Origine Nerveuse,' by Dr. H. C. Leloir, one of the Paris Theses; 'des Lésions Trophiques consécutives aux Maladies du Système Nerveux,' by Dr. X. Arnozan, another Thesis; 'Nature de l'Influence de l'Innervation sur la Nutrition des Tissus,' by Dr. E. Lahousse,'—a prize essay of the Royal Academy of Medicine of Belgium; 'Injuries of Nerves,' by Weir Mitchell; 'Maladie du Système Nerveux,' by Charcot; 'Diseases of the Spinal Cord,' by Erb, in Ziemssen, 'Trophoneurosen,' by Samuel, in Eulenburg's 'Encyclopædia,' &c.

Schwimmer and Leloir occupy themselves chiefly with the view of proving that certain diseases of the skin are dependent more or less directly upon the nervous system. Leloir's is an essay setting forth observations, mainly original, on the changes in the cutaneous nerves in certain diseases of the skin, which we shall consider presently, together with a review of the clinical facts which support the nervous origin of these diseases. Schwimmer's is a more comprehensive treatise, and is the most recent; and while in his zeal he has included some skin affections whose nervous origin will scarcely be accepted by most authorities, his careful and elaborate work most ably sets forth all the facts, cases and arguments that can be adduced in favour of this view, and he has brought together in an accessible form a large number of valuable cases that were scattered over the immense range of medical literature, and has also contributed many important cases from his own practice. Future workers in this field will therefore find their labour much simplified.

Schwimmer and Leloir have considered the subject from the skin side; I propose to take it up from the nervous system end, chiefly, but not exclusively, limiting myself to cases where definite lesions of the nervous system have been anatomically established.

While there are a large number of cases upon record in which lesions of the cerebrum have been associated with cutaneous eruptions, it is rare to be able to exclude the possible influence of the cord or other parts of the nervous system; moreover there are not a few instances in which the brain has evidently played an important part in the production of the eruption, and yet no anatomical change is likely to be present. Such are cases of disease of the skin due to mental emotion, as in alteration in the distribution of pigment, e.g. sudden blanching of the hair from fear, anxiety, or grief; though these are well known and authenticated, a case occurring under medical supervision is of interest. Eulenburg[1] and Landois had a patient in the hospital, in whom greyness of the hair and beard appeared in the night from excessive terror, while suffering from delirium tremens. Many instances of pigmentation changes, the result of mental emotion in other parts of the body, are on record. The frequency of vitiligo in the insane, and among epileptics, might be cited as evidence that the brain exercises an important influence upon pigmentation, both as to increase and decrease, doubtless through the sympathetic centre; but this influence is too vague for our present purpose. More to the point, but still of uncertain interpretation, is Bourneville and Poirier's[2] case of partial discoloration of the skin, in association with a tumour of the fronto-parietal lobe of the left side. Eruptions are frequent in general paralytics, especially pemphigoid bullæ. Déjérine records one where, twelve days before death, pemphigus broke out on the extremities, and, post-mortem, the nerves underneath the bullæ were found degenerated; there was also sclerosis of the lateral columns, as well as diffuse peri-encephalitis. It is impossible here to lay all the blame on the encephalitis, on the one hand, nor on the peripheral nerves, as Leloir appears inclined to do, and all analogy resists the assumption that the lateral columns were responsible. Indeed, if the observer were not so careful and skilful as Déjérine, one would suspect that some degeneration of the posterior columns had been overlooked. This case is cited as an example of how complicated the problem is as regards the cerebrum. Moreover, that these eruptions are generally due to the cord rather than the brain, is suggested by the fact that the bullæ, so Dr. Savage tells me, are always on the lower extremities in general paralytics, while the frequency of posterior sclerosis in them is well known. A somewhat more definite connection existed in a general paralytic I saw at Bethlehem, with Dr. Savage, in whom melancholia alternated with excitement. In the melancholic state his skin was cold, clammy, and intensely livid, but became normal during exaltation. Even with regard to vitiligo the cerebrum shares only an influence with other parts; thus, Bulkley,[1] confirmed by Debove and Barthélémy, speaks of the frequency of vitiligo in ataxics; and Leloir gives cases of peripheral nerve-changes in vitiligo patches when the presumption was against cord disease, though a negative was not anatomically proved.[2] But the loss of brain influence may affect the distribution of an eruption. Thus Janin de Saint-Just[3] records a case of a hemiplegic in whom scarlatina supervened; for two days the eruption was limited to the healthy side, and when it did attack the paralysed side, was pale and fugacious.

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[1] 'Archiv für Path.-Anat.' Bd. xxxv. p. 575.

[2] 'Progrès Médical,' 1879. Quoted by Leloir, p. 38.

Chevalier,[4] on the other hand, saw a case of small-pox, in which the eruption was confluent and hæmorrhagic on the incompletely paralysed side, and discrete and normal on the healthy side. Bouilly and Mathieu's[1] observation is appropriate in this connection. Intercurrent small-pox occurred in a patient after section of the sciatic nerve and the non-innervated limb remained free from eruption. These apparently conflicting facts are not so inexplicable as they would seem to be at first sight. In the third case there was total absence of all innervation, and no eruption. In the first, we may fairly suppose that there was slight vaso-motor influence, but no cerebral, and slight eruption; while in the second, the vaso-motor system was probably undamaged, but cerebral influence cut off, and the eruption was therefore violent. If this explanation be considered feasible, the eruption would be in inverse proportion to the brain influence. May not some such hypothesis as this afford an explanation for the varying severity of the eruption in the exanthemata? In confluent small-pox, for instance, the cerebral depression may be so great that the vaso-motor system is quite uncontrolled; whilst in the most malignant form this system is also paralysed, and death occurs, with no eruption except those from hæmorrhages into the cutis.

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[1] 'Archives of Dermatology,' 1878.

[2] Leloir, loc. cit. p. 33, &c.

[3] Quoted by Sané, Dict. Encyclop., tome vii. Series III. p. 307—article "Scarlatine."

[4] 'Développement des Erupt. Cutan. chez les Neuropathics.' Thèse de Paris, 1878.

Cases of herpes zoster on the paralysed limbs of hemiplegics are on record, such as that of Duncan,[2] in whose patient zoster appeared on the thigh, and almost at the same time hemiplegia of the same side. Payne[3] gives the case of a child who had zoster of the superficial branches of the anterior crural three days after the occurrence of a hemiplegia of the same side. Charcot,[4] who quotes these cases, warns us not to draw deductions too hastily from this coincidence, by relating the case of a soldier with ulcerative endocarditis, who had a sudden paralysis of one leg and at the same time zoster of the peroneal nerve-branches of the same leg. Post-mortem, there was an embolus in the posterior cerebral artery with its corresponding area of softening, and also a clot in one of the spinal branches of the lateral sacral artery, which pressed upon one of the posterior spinal roots of the cauda equina as the vessel went through a foramen. Oppolzer, Hessefink and Chvostek, and others, also give examples of zoster with cerebral lesions.

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[1] 'Archives Générales de Médecine,' 1880.

[2] 'Journal of Cutan. Medicine,' Oct. 1869, p. 69.

[3] 'British Med. Journal,' Aug. 1871.

[4] Charcot, loc. cit. p. 82.

A review of all the above facts suggests that the relation of the cerebrum to cutaneous eruptions is mainly due to the degree of inhibiting effect exercised upon the vaso-motor centre, but there are no facts indicating from what part or parts this influence starts in the brain to the vaso-motor centre.

When we come to lesions of the spinal cord in relation to skin eruptions, not only is the material more abundant, but our knowledge is somewhat more definite, though there is still much to clear up. The difficulty here, as in the cerebrum, is to find uncomplicated cases. The nearest approach to this are three cases, in which, with bullous eruptions, the columns of Goll were especially implicated. Of these I will give abstracts. The first two were under Schwimmer's observation; the third under Dr. Meyer of Strassburg, a full abstract of which will be published in the next number of 'BRAIN.'

The first one is described as Case 13, p. 148, of Schwimmer's work, and was that of a woman, æt. 32, who five months before admission had dyspepsia, followed by pemphigus, which only lasted a short time. In her present attack the whole body, face and scalp were involved in the pemphigus, which was hæmorrhagic on the legs; diarrhœa and albuminuria set in, and led to her death. At the post-mortem by Dr. Babes there was found sclerosis of the columns of Goll; in the cervical region, the peripheral part of the postero-lateral columns were especially wasted, and there was slight cell-increase round the vessels of the posterior horns or roots, and of the postero-lateral columns. In the upper dorsal region, the right anterior horn and the external nerve-cell group was atrophied. In the upper lumbar region, of which a figure is given, the whole periphery of the cord was atrophied, the posterior roots stained strongly, and the *columns of Goll sclerosed.* The nerve elements had almost entirely disappeared, principally from a wide-spread rich-celled vascular network. In the postero-lateral columns also the neuroglia was somewhat increased.

In the second case, No. 14 of his work, a previously healthy man had a severe chill from standing in water for some time. Soon after, the patient complained of pain and convulsive movements of the legs; he had the girdle-feeling, and then the bullous eruption broke out and soon developed into a pemphigus foliaceus. There was a temporary improvement, then a partial paraplegia came on, painful neuralgia of the upper limbs, bedsore on sacrum, and he died of exhaustion six months after admission.

Post-mortem, in this as in previous case, the cord was notably softened.

There was extreme sclerosis of the columns of Goll in the upper part of the cord up to the cervical enlargement, with neuroglia thickening. At the beginning of the dorsal region circumscribed, greyish, softened gelatinous places existed at the anterior periphery, especially the left, and the anterior horn was atrophied. At the beginning of the lumbar part there was widespread and extreme sclerosis of the posterior root-zones, with thickening and cell-increase of the vascular network, and atrophied foci of the left anterior horn corresponding with the median cell group.

Meyer's case was that of a man of 65, with previous good health. The disease began as an eczema, then it spread nearly all over the body; bullæ appeared, diarrhœa set in, and he died seven weeks from the outset of the skin eruption. Post-mortem, there was degenerative parenchymatous neuritis of many of the cutaneous nerves, sclerosis of the columns of Goll in the dorsal region, with points of sclerosis scattered through the posterior columns in the upper cervical and in the lateral columns of the dorsal and lumbar regions. The neuroglia was increased, and the walls of the blood-vessels thickened.

Scarcely less interesting, though there was no post-mortem, was Case 12 of Schwimmer, a private patient, a man aged 64, who in a street accident sustained a severe bruise of the left shoulder. After a fortnight's severe pain of the whole extremity it left a neuralgia of the upper limb, which came on at irregular intervals; three weeks later, a severe herpes zoster affected the whole extensor aspect of the limb from the shoulder to the ends of the fingers; the neuralgia gradually diminished, and then bullæ appeared, first on the left leg and then on the trunk, coming out in crops, and attended with a rise of temperature. For the first ten days the bullæ were limited to the left side of the body, then spread to the right side, and soon became universal, including the oral mucous membranes. Abundant crops of semi-opaque bullæ, preceded by severe pain, exhausted the patient and produced great denudation of epidermis. On the sixth week of the pemphigus outbreak, paraplegia set in, with paralysis of bladder and rectum, and the patient died three months from the commencement of the herpes zoster. No post-mortem was allowed.

Interesting as these cases are, while showing that there is an association of posterior sclerosis with some cases of pemphigus, they do not even prove that the cord lesion was primary, though it was probably the case. Meyer suggests for his that the vascular obliteration produced sclerosis of the cord, and the same change in the skin-vessels predisposed the skin also to disease.

In the fourth case there can be little doubt that there was neuritis from the injury, and that the inflammation travelled up to the cord, and spread in the cord and led to the bullous eruption.

There is no disease of the cord in which eruptions are so frequent and in such great variety as in locomotor ataxy; but very often the lesions are not confined to the posterior columns, by the time they come to the post-mortem table, but there are some such in whom there have been eruptions during life. I will first enumerate the eruptions that have been noticed in ataxy:

1. Transitory erythema. { (*a*) Simple.
   (*b*) Nodosum.
2. Urticaria, with intense itching.
3. Papular or "Lichenoid" eruptions, probably eczematous.
4. Ordinary eczema.
5. Herpes zoster.
6. Pemphigus.
7. Pustules "like ecthyma."
8. Ulcers, superficial and deep.
9. Superficial gangrene.

10. Perforating ulcer of foot.
11. Falling off of the big toe-nail.
12. Leucoderma.
13. Petechiæ and ecchymoses.
14. Unilateral sweating.
15. Œdema.

The first nine of these lesions occur in the early stage. They are generally associated with an exacerbation of the lightning-pains, and are usually limited to the course of the nerves in whose course the pains are felt.

The Erythema, besides being transitory, is uncertain in size and position, and may be anywhere; it is especially alluded to by Charcot.[1] Erythema nodosum is mentioned by Bristowe.[2] Déjérine, Purdon, Friedrich, &c., describe urticaria. Charcot[3] had a case of a woman in whom enormous plateaux of urticaria were produced at the most painful spots during an attack of pains. In another, the right buttock was covered with herpes zoster in the course of the lightning-pains. In a third, an old woman, in whom the ataxy ran a very rapid course, two unusually severe attacks of lightning-pains were associated with very numerous pustules, "like ecthyma," which led to deep ulcerations in the course of the small sciatic and left saphena-nerve respectively; and in the first attack there was also a deep *slough*, 5 centimetres across, on the right sacral region, which took two months to heal.

The papular or lichenoid eruptions are especially common, and more or less confluent upon the legs and thighs; but may be restricted to one leg, or even to the course of a single nerve. Charcot does not appear to believe in an ordinary vesicular eczema in ataxy; but Purdon[4] found long-standing eczema of the lower extremities in the latter part of the course of ataxy; and inasmuch as several cases of eczema following neuralgia, limited to the painful area, such as that of Cavafy,[5] are known, there seems every *à priori* reason that such eczema may occur sometimes and bear relation to the spinal lesion; at the same time eczema is so common, that the connection between the two must not be hastily assumed because they are present together. Pemphigus, in cases of chronic myelitis and acute meningitis, is very common. Vulpian[1] records a case with *ataxy* and lightning-pains, in whom bullæ appeared on the painful upper limbs whenever he put his hands in hot or cold water, and even spontaneously on some occasions; subsequently an elephantiasis-like condition was developed in the hands, and the finger-nails were much deformed. Gangrene is rare in locomotor ataxy; but in addition to the case of Charcot's already cited, Debove[2] records a case of a woman, æt. 44, with ataxy and lightning-pains, in whom a slough formed on the upper part of the sacrum which spread to the left buttock, and got deeper; then invaded the right buttock, and became symmetrically developed on each side of the median line, with an area of 8–9 by 6 centimetres, accompanied by *œdema* of the lower limbs. Post-mortem, there was atrophy of the posterior roots, especially at the lumbar enlargement, and sclerosis of radicular fasciculus; and in the nerves, on the border of the ulcer, parenchymatous neuritis. Perforating ulcer of the foot is now well known to be sometimes due to locomotor ataxy, although it may also be due to lesions of the peripheral ends of the nerves, the nerve-trunks, or the spinal ganglia of the affected side. Hanot and many others have demonstrated this. Falling-off of the big toe-nail occurs in ataxy, occasionally without any preceding injury, or inflammation; in one case[3] it occurred three times within eighteen months; regeneration occurs without any difficulty.

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[1] Charcot, loc. cit. p. 76, &c.

[2] Bristowe's 'Medicine,' 3rd edition, p. 912.

[3] Charcot, loc. cit. p. 65.

[4] Purdon, 'Union Médicale,' 1878, No. 75.

[5] Cavafy, 'Brit. Med. Jour.,' July 24, 1880.

Leucoderma, as already mentioned, has been noted in connection with ataxy by Bulkley, Debove and Barthélemy.

Unilateral sweating has recently been recorded by Raynaud and Artaud.[4] It occurred on the right side of the face, head, neck and shoulder ten or fifteen minutes after a meal, with no alteration of the pupil, but there was an increased flow of saliva on the affected side. Œdema has already been alluded to in Debove's case; it is especially apt to occur in the knee and ankle joint, as mentioned by Schwimmer,[1] and often disappears spontaneously.

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[1] Vulpian, 'Maladies de la Moelle,' 1877, p. 57.

[2] 'Progrès Médical,' quoted by Leloir, p. 115.

[3] Joffroy and Pitres, 'Progrès Médical,' 1882, No. 8.

[4] 'Revue de Médecine,' 1884.

Only one reference to the cases illustrating each cutaneous lesion has been given; but the number could, with regard to most eruptions, be easily multiplied.

The great variety of eruptions, with apparently the same kind of nervous lesion, is most striking, and we can neither predict the kind of eruption from the nature and position of the lesion, nor, with but few exceptions, can we predict the position or nature of the lesion from the kind of eruption alone. At the same time it must ever be borne in mind that, common as some kind of eruption is in locomotor ataxy, in the majority of cases with the same lesion there is no eruption at all; still the frequency is too great for the association to be purely accidental, and we can only infer that, besides the cord lesion that can be proved to exist, there are some other conditions that determine the occurrence and the nature of the eruption. Of these conditions at present next to nothing is known. The coincidence of the majority of the eruptions with lightning-pains suggested that if the position of the lesion producing them can be ascertained, that for the eruptions will also be there or thereabouts; and this, as Charcot shows, has been pretty accurately ascertained by post-mortem proof that where lightning-pains had been present, there was always sclerosis of the posterior columns; while the other lesions which might possibly account for them were absent in different instances, viz. atrophy of the posterior roots, posterior spinal meningitis, sclerosis of the posterior cornua, and irritative lesions of the spinal ganglia or peripheral nerves. A most striking instance is a case of Charcot's,[2] in which a woman had suffered severely from lightning-pains for fifteen years, but with no motor symptoms; at the autopsy the only appreciable lesions were in the posterior columns, where there was commencing sclerosis, with no alteration of the nerve-tubes, imbedded in the thickened neuroglia.

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[1] Loc. cit. p. 181.

[2] Loc. cit p. 78.

The posterior root zones, Charcot thinks, consist of sensory fibres, a certain number for the performance of reflex acts, and others which have a more or less direct influence on the nutrition of the skin. These last, he suggests, are less easily irritated than the sensory fibres, and this may account for the frequent absence of eruptions, on the one hand, and for their varying intensity where present, and so for the existence of erythematous, papular, vesicular, bullous, pustular or gangrenous eruptions. It must, however, be borne in mind that the last three eruptions are also frequently the result of the loss of nerve influence, rendering the tissues unable to resist noxious external influences. While therefore a rather strong case is made out in favour of the postero-external column being largely concerned in ataxic, and many other skin eruptions due to cord disease, we cannot say that they are the sole arbiters in this matter. The cases first related point to the columns of Goll having some influence; of the posterior grey cornua we can say nothing; of the antero-lateral columns and anterior grey cornua, the evidence is against them for the most part. The few cases in which there have been eruptions in association with diseases in which the anterior part of the cord is mainly concerned, have been always open to the suspicion of their being more or less complicated. Thus Balmer [1] gives three cases of progressive muscular atrophy with slight loss of sensibility, in the course of which pemphigus supervened.

If Gowers's [2] supposition, that there are some sensory fibres in the tract in front of the crossed pyramidal tract, as supported to some extent by animal experiments of Woroschiloff and Ott, and by Gowers's own case and that of Hadden [2] (in the last, unfortunately, the clinical history is unknown), this column may have to be considered with regard to a possible influence on the skin; nevertheless it remains that the posterior columns of the cord have a very important influence upon the nutrition of the skin; and the more or less direct production of cutaneous eruptions. Before concluding the consideration of the spinal-cord influence on the skin, mention must be made of the numerous eruptions recorded in association with spinal meningitis, especially vesicular eruptions, such as the herpetic and pemphigoid. Erb[1] also enumerates, roseola, petechiæ, erythema, urticaria and erysipelas. With the exception of erysipelas, which was probably either a purely accidental concurrence, or an error of diagnosis on the part of the recorder, all the lesions are similar to those found in locomotor ataxy, and they are doubtless explicable by the irritation set up in the posterior columns, on the one hand, or in the issuing nerves, on the other. Erb also states that herpes and bullæ are frequent in slow compression of the cord.[2] As in the brain, vaso-motor disturbances may be set up in connection with cord disease without any visible lesion. Thus in acute ascending paralysis, in which lesions have hitherto been searched for in vain,[3] Eisenlohr had a case with transitory œdema, redness over the joints and profuse sweating.

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[1] 'Archiv für Heilkunde,' 1875, p. 317.

[2] 'Diagnosis of Diseases of the Spinal Cord,' pp. 13 and 14, 1st ed.

The lesion for acute decubitus cannot be localised more definitely than by saying it is found especially in acute central myelitis; and that it may occur in ataxy has already been shown. Its occurrence in cerebral lesions, hæmorrhagic, softening and traumatic, is well known, and it is then always on the paralysed side, and pressure, though often very slight, seems to determine its position.[4] Brown-Séquard has shown that in two-thirds of the cases the cerebral lesion is on the right side. I am not aware of any case being recorded showing that the cord was free from secondary change in such cases.

Coming now to the cerebral and spinal-nerve ganglia, the ground is somewhat firmer. It was here that Bärensprung first demonstrated anatomically, what had long been urged on clinical grounds by Rayer, Charcot, Parrot and Danielsen,[5] the neurotic origin of herpes zoster, and while it was the first to be thus proved, it is also the type of neurotic dermatonoses. Bärensprung demonstrated that in his case there was interstitial neuritis of the spinal ganglion, and of the trunk of the nerve coming from it. This observation has been abundantly confirmed for the majority of cases by many subsequent observers, and the presence of the same kind of lesion, namely, inflammation of the Gasserian ganglion,[1] has been proved in cases of herpes frontalis; but Bärensprung's assumption that all cases of zoster were dependent on lesion of the nerve-ganglion is now known to be true for the majority of cases only, and not for all.

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[1] 'Pathological Society's Transactions,' vol. xxxiii., p. 21.

[2] Ziemssen, vol. xiii. art. "Spinal Meningitis."

[3] Quoted by Erb in Ziemssen, loc. cit.

[4] Quoted by Bastian, 'On Paralysis from Brain Disease,' p. 210.

[5] Danielsen, it must in justice be said, had proved a neuritis of two intercostal nerves in a case of zoster, before Bärensprung showed that the ganglia were at fault.

As already mentioned, herpes is common in cord disease, especially central myelitis, ataxy, and meningitis, and in a few instances the ganglion has been examined and found to be quite normal. Werdner[2] in one case found a lesion of the posterior spinal roots, while the cord and spinal ganglion were healthy. Strangely enough, the posterior roots are healthy as a rule when the ganglia are affected, which is contrary to what would *à priori* be anticipated.

The cases of zoster with cerebral lesions have been alluded to, and they are too open to fallacy to argue from. There are also cases which give clinical evidence that the ganglia are not always at fault; thus cases of double zoster are very unlikely to be due to inflammation of both ganglia, and as many are of syphilitic origin, probably a local meningitis would account better for them.

There would even seem to be a possibility of some cases being due to reflex irritation. Jewel[3] relates a case of zoster cruralis as a consequence of severe uterine disease, the zoster and neuralgia disappearing when the uterine trouble was removed. Lendel[4] relates cases of zoster from poisoning by carbonic oxide gas, which he ascribes to a neuritis toxica, while Schwimmer regards them as reflex. Injuries and neuritis of the trunk of the nerve have frequently given rise to zoster, and while it is very likely that the zoster was directly due to this neuritis, the ganglia have never yet been proved to be normal in such cases. The periphery of the nerves at the skin has not been examined, but the presence of defects of sensibility in some cases is suggestive that the cutaneous nerves may be affected, at all events.

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[1] By O. Wyss and Horner, quoted by Leloir, p. 152.

[2] Quoted by Leloir, p. 154.

[3] 'Trans. Amer. Neurological Assoc.,' Nov. 1875.

[4] Lendel, 'Archives Génér. de Médecine,' 1865.

There is no doubt that peripheral irritation will produce herpes. I have seen a case in which inflammation at the root of a tooth, leading to abscess and a sinus, excited severe attacks of herpes round the sinus, which ceased when the tooth was removed. Mr. Pearce Gould had a case of caries of a rib, with recurrent herpes. Lesser[1] relates a case of a young man who had the nerve destroyed of the last molar but one in the lower jaw. Two days after, groups of vesicles, accompanied with pain, appeared on the chin and lower lip in the course of the left mental nerve; five days later the first crop had dried up, but three fresh groups of vesicles came out on the left cheek and one vesicle on the left auricle. Sensibility was normal. Lesser explains this as follows: inflammation spread from the branch of the alveolar nerve to that nerve itself; thence to the mental nerve, which is a direct continuation of the alveolar nerve; then advanced centripetally, and then down again, affecting the auricular temporal nerve. If this explanation be true, and it seems the most probable, the eruption in the tract of the mental nerve was produced by an inflammation of the nerve without the implication of the ganglion; and in my case, and that of Mr. Gould's, this seems also have been the case: anatomical proof of this cause of herpes without implication of the ganglion is still wanting. The lesion of the ganglion is not always the same. Kaposi[2] had a case of herpes frontalis, in which he found hæmorrhage into the Gasserian ganglion. Wyss and Satter have had similar cases. Hæmorrhage into the cauda equina with crural herpes has also occurred. Charcot's case of herpes from an embolus in a branch of a sacral artery has already been mentioned. He also relates a case of carcinoma of the vertebræ with herpes; while Ollivier[3] found cancer of the pleura and lung on the same side as the herpes.

Gunshot and other injuries, as related by Weir Mitchell,

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[1] Virchow's 'Archiv,' vol. xciii. 1883.

[2] Kaposi, 'Hautkrankheiten,' p. 322, 1883.

[3] 'Wiener Med. Wochenschrift,' 1877, No. 25-27.

Morehouse and Keen, &c., are other causes of neuritis only, as the eruption was always some time after the injury; herpes may thus occur as a result of any kind of irritant in the ganglion or any part of the issuing nerve. It has been anatomically proved that the lesion may be either in the cord, the posterior root, or the ganglion, and it is probable that a lesion of the cutaneous nerves alone is capable of producing it. Reflex herpes is of more doubtful existence. It has never been shown that it may occur from uncomplicated cerebral lesions, those cases associated with cerebral lesions being always more or less open to the possibility of other nerve defects.

Similar instances of the same eruption being due to lesion of different parts of the nervous chain are seen in pemphigus leucoderma, and other pigmentary alterations, in perforating ulcer, &c.

Cutaneous disease from lesions of the branch or branches of nerves below the ganglion have been especially described by Paget, Weir Mitchell, Morehouse and Keen, and their observations have been confirmed with very little addition by many others. Leprosy also, by its implication of the nerves, affords us information in the same direction; but even here, as in more central disease, the total absence of defects in the nutrition of the skin where the same kind of lesion in the nerves existed is quite as striking as the occurrence of the eruptions. The following observations are mainly from Mitchell's well-known work. The nutritive and other defects that have been recorded are:—

1. Erythema, persistent and abscess-like.
2. Herpes.
3. Bullæ.
4. Ulcerations. { Simple.
   Perforating.
5. Eczema.
6. Glossy skin.
7. Defects of nails.
8. „ hair.
9. „ pigment.
10. Chronic œdema, like Elephantiasis.
11. Ichthyosis-like condition.

In complete division of a nerve, there is with the general atrophy "œdema, with finally discoloration of the dry and thickened skin, the ragged epithelia hanging in patches."[1] Some ulcerations also occur, but these are the result of injury, often slight, which the non-innervated tissues are unable to resist. Bullæ are rare in complete section of a nerve. Mitchell never saw them in the small number of cases where there was section of all the nerves supplying a part; yet, as Arnozan points out, the bullæ come where the anæsthesia is complete. Nevertheless, bullæ do come out where the function of the nerve is quite destroyed; as in cases of anæsthetic leprosy, where both paralysis and anæsthesia are complete, bullæ come out from time to time on exposure to cold. Like the ulcers, therefore, they require a local irritation to produce them. These bullæ often leave slow-healing ulcers; yet, as a rule, after total section of a nerve, wounds heal fairly well, if put in proper condition. Where the nerves have been injured in a slight degree, the eruptions are much more numerous and important. The erythema that is sequential to nerve injury lasts long and simulates an abscess. This form of erythema is regarded by Paget[2] as characteristic of nerve injury, a statement which is confirmed by Charcot and Brown-Séquard. Probably this is the same condition that is described by other authors as erythema nodosum, which occurs in cases of neuritis. Herpetic eruptions are frequent after injury to nerves. Brown-Séquard,[3] mentions a contusion of the internal cutaneous nerve followed by herpes in the affected region. In a healing wound over an injured median nerve, Mitchell witnessed ulcerated matrices of nails and vesicular eruption. There is also Rayer's[4] well-known case, reported by Charcot, of gunshot-wound of the thigh, followed by neuralgia and the outbreak of repeated crops of herpes at the painful spots. These cases could easily be multiplied.

Bullæ are on much the same footing as herpes. In one of Charcot's[5] cases, abscesses and the necessary incisions injured the nerves of the arm, and was followed by neuralgia and other symptoms; with bullæ, which formed in a few hours, grew as large as a nut, and left a quickly healing ulcer. These came chiefly about the joints of the first three fingers. Mitchell quotes several other cases in the same place of this kind of lesion.

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[1] Weir Mitchell, loc. cit., p. 152.

[2] Paget, 'Med. Times and Gaz.' 1874.

[3] Weir Mitchell, loc. cit. 153, quotes this.

[4] Ibid.

[5] Ibid.

In non-tuberculated leprosy, among the early symptoms are shooting-pains, with the eruption of bullæ upon the fingers and toes. These differ from the bullæ of the advanced anæsthetic leprosy in being spontaneous, small, numerous, and occurring on a painful surface; while the others are large, solitary, on an anæsthetic part, and usually excited by local causes.

Ulceration generally follows the bullæ in partial injury to a nerve, and is not due to local injuries like ulceration after total section; sometimes, as Hutchinson states, it begins with a whitlow-like condition, with considerable inflammation. The whitlow is painful or not, according to the locality in an anæsthetic or hyperæsthetic area. Mitchell[1] also describes eczematous conditions either on healthy or atrophied skin. Cavafy's already cited case confirms this; and Arnozan relates a case of Brouardel's, where a man of 60 had a contusion of the shoulder, followed by pains shooting down the arm, and in forty hours an eczema, limited to the radial nerve, becoming rapidly confluent and getting well in a fortnight.

The condition known as "glossy skin" appears to be always the result of neuritis, traumatic, gouty, or rheumatic, &c. It begins some little time after an injury, and is often excited by an inflammation supervening on the original wound. Thus Mitchell relates the case of an attack of erysipelas supervening upon a wound of the radial, followed by the peculiar "causalgia," glossy skin and eruptions; the patient having previously had partial loss of motion and sensation. It is in glossy skin that eczema is so frequent and often very persistent.

Mitchell gives a case where the glossy skin was associated with an antero-lateral sclerosis; intense neuralgia, motor palsy of legs, with contracted toes and ankles; no loss of sensation; neuralgia of arms and hands; contraction of fingers; no palsy of upper limbs; hyperæsthesia of palms; causalgia and glazed redness of ulnar side of palms. It is evident that there were other secondary changes besides the antero-sclerosis, but whether it extended down the nerves of the limbs cannot be determined. There were nutritive changes in the nails and teeth in this case.

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[1] Loc. cit., p. 154.

"After total section of a nerve the nails are apt to become clubbed, and in rare cases to suffer from painless whitlows" (Mitchell):[1] growth is also retarded, but not stopped, and nails may even be reproduced. Furrowing and white spots are common.

In partial destruction of a nerve, painful ulceration of the matrix may occur; and when there is glossy skin, the nails curve both longitudinally and transversely with thickening of the cutis beneath; or else the line of union of the nail and skin begins lower down under the nail, and the border is ragged and notched. In the toes the nails are less deformed, but a painful and recurring ulceration occurs at their angles.

In cases without glossy skin, the nails may be only dry, scaly, very thin, cracked and fragile; or furrowed, pitted and discoloured. In hemiplegias nail-growth is arrested.

The hair falls off, or is very scanty and poor in glossy skin; in neuralgias and injuries of the trigeminal it often becomes white or falls out temporarily; in some cases the blanching persists. In a traumatic neuralgia, Ponteau saw the hair grow large and hard, with a tendency to stand erect.

Larrey describes similar cases on hyperæsthetic skin.

Crampton witnessed a case in which the arm became thickly hairy in the bend of the elbow after a lancet wound[2] which injured the musculo-cutaneus nerve in "bleeding," and is supposed to have set up a neuritis. Section of the nerve gave partial relief, but the skin recovered after salivation for an attack of pneumonia.

Schwimmer[2] gives a case of a wound of the temple, followed by an alopecia patch in the temple and upper lip of the same side.

In general, there is loss of hair after division of a sensory nerve, and increase after division of the sympathetic.

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[1] Hutchinson, quoted by Mitchell, p. 153.

[2] Schwimmer, loc. cit. p. 222. He also quotes the other cases from Mitchell.

With regard to pigmentation defects, Schwimmer[1] gives a case of bilateral herpes in which in the course of the affected nerves white patches appeared; while he quotes O. Wyss's case of loss of pigment from disease of the second division of the trigeminal.

In another of Schwimmer's cases after the herpes, spots and striæ appeared on the back of the forearm in lines to the back of the hand and fingers. The ganglia may have been implicated in these cases.

A chronic œdema producing an elephantiasis-like condition is sometimes induced.

Mitchell gives the case of a bullet-wound of the arm near the axilla; there were darting-pains down the limb; the skin was dry, scaly, and yellow, and an elephantiasis-like condition of the thumb and first two fingers and the radial side of the back of the hand; there was slight œdema of the ulnar side of the palm and back of the hand.

An ichthyotic condition is occasionally developed after an injury to a nerve. Leloir gives a case that was under M. Trélat.—A man fell and received a contused wound, dividing the ulnar nerve incompletely; the ulnar region became anæsthetic. This lasted a short time, but during that period the affected region was in an ichthyotic condition; the skin was thickened and peeled, and looked like "serpentine ichthyosis." As sensation returned, the skin improved, and three weeks after the injury, sensation and the skin were normal.

In another case this condition supervened after a wound of the nerve of the index-finger. There was recovery in a short time of sensation and health to the skin. In paraplegics this condition also occurs. Leloir gives cases, and figures one with ichthyosis of the legs in a painter, who suffered from lead-poisoning and cerebro-spinal disease. The cerebral arteries were thickened and atheromatous. The spinal cord was not examined.

Congenital cases of ichthyosis hystrix in the course of nerves are well known, but they appear to be in a somewhat different category to ordinary ichthyosis.

These cases of acquired ichthyosis probably differ from the ordinary congenital form, but there is no other term that would so well convey the appearance.

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[1] Schwimmer, loc. cit. p. 211. Leloir, loc. cit. p. 59.

That perforating ulcer results from atrophic degenerative associated with interstitial neuritis, has been established by many observers. Poncet,[1] Duplay[2] and Moralt, Michaud, Sonnemburg[3] and Savory,[4] and Butlin have all made valuable contributions in this direction. In anæsthetic leprosy it is common, and Déjérine and Leloir have made researches upon this subject.

The ganglia and the peripheral nerves have also been found in a condition of atrophic parenchymatous neuritis in perforating ulcer.

The original part of Leloir's Thesis is mainly concerned with the lesions of the cutaneous nerves. To go fully into the subject would be, but to reproduce his very careful and valuable work.

That there should be lesions of the cutaneous nerves in eruptions of the skin at the site of the eruption might be anticipated as a probable event, the nerves being involved secondarily to the skin lesion; but research shows that the nerves are not so easily affected, and when therefore they are found to be diseased, it is the more significant. Déjérine and Leloir have examined four cases of psoriasis, three of tubercular lupus, one of lupus erythematosus, five of ecthyma, (three of them occurring in typhoid fever, and two in cachectic individuals), a case of ulceration of the big toe, with melanodermia and epithelioma of the lip, and all with negative results. Balzer has never found lesions of nerves in syphilides, psoriasis, lupus, or epitheliomas; Chambard has not in gummata; Poncel in the neighbourhood of abscesses; and Quénu where the pneumogastric traversed cheesy glands; nor Vulpian in gangrene. Weir Mitchell, Cornil, and Ranvier, &c., confirm these observations.

Leloir therefore considers that he is entitled to assume, that when cutaneous lesions are found they are primary.

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[1] 'Mémoires de Médecine Militaire,' 1864, tome xii.

[2] 'Archives de Médecine,' 1872.

[3] Sonnemburg, 'Deutsche Zeitschrift für Chirurgie,' 1876.

[4] 'Medico-Chirurgical Trans.,' 1879, p. 360, &c.

In all, the character of the lesions is the same, viz. atrophic parenchymatous neuritis.

The nerves were found affected thus in three cases of vitiligo; two by Leloir, one by Déjérine.

In one of these there was syphilis, but no central lesion proved or probable on clinical grounds.

The second and third cases were fatal cases of phthisis. Here also no central lesion was to be anticipated, but in none of them was there any examination of any other parts of the nervous system.

Lesions were found in two cases of ecthyma. One was a general paralytic; the other with cerebral hæmorrhage on the right side, and the ecthyma on the left side.

In one serpentine ichthyosis case, pemphigus was also present, and the centres were not examined.

In three cases of pemphigus—

1. Examined by Déjérine, has already been alluded to. The patient was a general paralytic, and peri-encephalitis and lateral sclerosis of cord were found

2. Examined by Quinquaud. There was spinal hemiplegia sclerosis of the lateral column encroaching upon the anterior and posterior columns.

3. By Leloir. A woman aged 72, very cachectic on admission to the hospital, died from exhaustion from "bilious vomiting." The centres were not examined.

4. Jarisch[1] also has found the cutaneous nerves diseased in a similar way in a woman who had so-called herpes iris. He also found changes in the central, lateral, and posterior parts of the cord.

Thus, out of the ten cases, five had certainly central disease. In the other five the centres were not examined. The complication of the ichthyosis with pemphigus reduces its value somewhat, and we cannot argue from it. In the case of the old woman who died with "bilious vomiting," the absence of central disease can scarcely be affirmed positively. This leaves the cases of vitiligo. In these there was probably no actual central lesion; though since we know that even mental disturbance will produce vitiligo, it would be rash to say that some central influence had nothing to do with it.

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[1] 'Vierteljahr. für Dermat. und Syph.' 1880.

In the other five cases it would seem probable, to say the least, that the central lesion had as much to do with the peripheral nerve lesion as that lesion could have with the eruption; and it is arguable that, though lesions of the skin do not affect the nerves of the part as a rule, they may do so when the protecting influence of the nervous centres is in abeyance, even if not acting adversely. But since we see that so many skin lesions are conduced to, if not actually produced by, a defective link in the nervous chain of brain cord, ganglia or trunk of a nerve, it seems probable that a defect at the peripheral end, viz. at the cutaneous nerves, might produce skin disease; but nevertheless, in the face of the above considerations, we must pause before we jump to the conclusion, that these lesions of the cutaneous nerves occurred independently and produced lesions of the skin.

There is, however, one case examined by Leloir and Déjérine which is less open to fallacy, and in favour of Leloir's contention.

In this, a woman, the subject of chronic rheumatism and muscular atrophy, with albumen in the urine, with dulled intellect and cachectic appearance, æt. 49, died twenty-three days after admission—five days before death three sloughs formed on the lower part of the sacrum, of the size of a crown. The cutaneous nerves were in a condition of parenchymatous neuritis in the neighbourhood of the slough, the intramuscular nerves were similarly affected, the spinal roots and the spinal cord throughout its whole length were quite healthy. The brain and meninges were quite healthy to the naked eye. Still the cerebral influence could scarcely have been healthily exercised. In two cases, one of a woman with hemiplegia, in which cutaneous gangrene followed the application of a mustard poultice; the other, of a cachectic woman attacked with cutaneous and visceral gangrene, Leloir found the cutaneous nerves were quite healthy. These give more force to the first case. In Leloir's Case XXX. of patches of cutaneous gangrene in a hysterical girl, had the case not been seen by such able observers as Hardy, Vulpian, and Lailler, I should certainly from the description have come to the conclusion that the lesions were self-induced and nervous only in reference to her mental condition.

His Case XXIX. has been already alluded to under gangrene with ataxy, and though the cutaneous nerves were affected, few will allow that they were primarily the cause of the sloughing, as Leloir contends.

Quinquaud, quoted by Percheron,[1] has published a case of general exfoliative dermatitis, with parenchymatous neuritis of the cutaneous nerves, supplied by the intercostal and sciatic with diffuse myelitis; and Lancereaux[2] has also published a similar case where the nerve centres are involved.

These facts certainly suggest the probability that the cutaneous nerves do not give way until the central influence is weakened.

Finally, the sympathetic and its lesions remain to be considered. Although the number of cases affording clinical evidence of the influence of the sympathetic upon the skin is considerable, there are but few cases where an actual lesion has been demonstrated, and even in some of these the proof is indirect.

The effects are shown in,

1. Congestion.
2. Pallor.
3. Pigmentation.
4. Sweating.
5. Increased growth of hair.

The close relationship of the sympathetic with the cervical enlargement of the cord is universally acknowledged, so that lesions here with vaso-motor phenomena may fairly be accepted as demonstrated lesions of the sympathetic. These two cases of Rendu[3] illustrate opposite effects.

The first was a case of dislocation of the 6th cervical. There was paraplegia; the pupils were contracted; the face was hot and turgid, although the skin of the face and neck were of a violet tint. In the cheeks and nose the smallest capillaries were visible, the ears were of a deep red; the rest of the body was normal.

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[1] Percheron, 'Thèse de Paris; dermatitis exfoliatrice.'

[2] Lancereaux, 'Traité d'Anatomie Pathologique,' tome i. p. 275.

[3] 'Archives générales de Médecine,' 1869, II.

In the second case there was a fracture of the two last cervical and the first dorsal vertebræ, with generally paralysis and extreme pallor of the whole surface of the skin.

Mitchell[1] gives a more direct case. The right sympathetic was wounded by a bullet. Among other symptoms the man had contracted pupil, with unilateral flushing of the right side of the face on exertion.

The erythematous and urticarial lesions that have so often been spoken of in relation to various nervous lesions are doubtless immediately due to the action or inaction of the sympathetic, but we have no more positive evidence of sympathetic defect in these cases. In a case of universal acute eczema, Marcacci[2] found changes in the sympathetic.

Such doubtful lesions as hyperæmia of cervical ganglia in unilateral sweating (Ebstein), pigmentation of ganglia in pemphigus, universal atrophy of the skin and sclerema (Schwimmer) do not count for much. Indeed, the same pigmentation was found in a healthy man killed by accident.

With regard to pigmentation, out of 33 cases of Addison's disease,[3] in 21 there were lesions of the abdominal sympathetic. The changes were partly from thickening and partly from atrophy, and often only affected single ganglia of the cœliac plexus. There has also been found fatty degeneration, caseous degeneration, small cell infiltration, in single ganglia, &c. The proof of the dependence of the pigmentation in Addison's disease upon the sympathetic is therefore pretty strong; the absence of pigmentation in some other cases where the ganglia were diseased being only in accordance with the other lesions of the nervous system and the skin. The influence of the sympathetic upon sweating is generally acknowledged.

In Traube's[4] case there was a tumour in the cord half an inch below the medulla oblongata, and profuse sweating a few days before death.

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[1] Loc. cit. p. 318.

[2] 'Giornale Italiano delle Malatie della pelle,' 1878, June number.

[3] Guttmann in Eulenburg's 'Real-Encyclopädie,' I. Bd. s. 128.

[4] 'Gesammelte Beiträge zur Path. u. Phys.,' Berlin 1871, ii. p. 1012.

Claude Bernard's experiments showed, that paralysis of the sympathetic ganglia produced hyperidrosis. The dependence of the sebaceous secretion upon the sympathetic is only inferrible from analogy; but Oscar Simon divided the nerve supplying the oil-gland of the duck, stimulated the gland end, and saw the oily droplets poured out.

The relation of the sympathetic to the sensory fibres is shown by various experiments and facts in regard to the induction of sweating.

Brown-Séquard produced sweating by the direct excitation of sensory nerves.

Kendall and Luchsinger[1] produced sweating in an amputated limb, twenty minutes after the operation, by irritating the nerves of the stump.

Dieffenbach found in an artificial nose that sweat returned when sensation was recovered. Whether the centre for the regulation of the sweat is in the medulla oblongata or cord is doubtful, but there is no doubt that the path of conduction is in the sympathetic.

The hair-growth is certainly influenced by the sympathetic; in rabbits, division of the cervical sympathetic is followed by increased growth of hair. The cases of injury to nerves affecting the hair already gone into, doubtless act through the sympathetic, but no lesion is provable.

The actual anatomical facts, therefore, with regard to the sympathetic are very meagre; but it is not by any means to be inferred that the sympathetic influence is not of high importance with regard to skin lesions, and indeed it is not improbable that the rest of the nervous system acts upon the skin almost entirely through the sympathetic.

The general statements that may be made from a consideration of the preceding facts are as follows:—

That less serious consequences ensue from cutting off the nervous supply than from irritant or inflammatory lesions of the parts of the nervous system that affect the skin.

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[1] 'Archiv für die gesammte Physiologie,' 1876, p. 212.

That the kind of eruption produced by the nervous system varies greatly, often without any evident reason, when the nervous defect is apparently the same in place and kind.

That the same eruption may owe its origin to any defective link in the nervous chain from the centre to the periphery.

That the same kind of nervous lesion that at one time appears to excite an eruption or other nutritive defect in the skin, even more frequently produces no change in the skin whatever.

The lesions other than atrophy that result when innervation is abolished are often traceable to external injurious influences which the tissues, when unprotected by the nervous system, are unable to resist; but we know nothing of the conditions that determine the nature of the eruption or other skin defect when the nerve lesion is irritative, nor what it is that determines whether there shall be any eruption, or none at all. This uncertainty of effect suggests that the nervous influence is an indirect one.

The cerebral effect appears to vary according to whether its control over the vaso-motor centre is increased or decreased and to the secondary changes it induces in the cord. No localising lesions have yet been found for its influence on the vaso-motor centre.

In the spinal cord the fibres that preside over the nutrition of the skin are bound up with the sensory fibres, and reside therefore mainly in the posterior columns.

That outside the cord the path is by the posterior roots, the spinal ganglia, and the sensory fibres, and that lesions of any one or more of these may lead to changes in the skin.

In drawing up this paper my aim has been to place before the readers of 'BRAIN,' in a small compass, the facts that pathology affords with regard to the relation of the nervous system to the skin, and to give a *résumé* of the most definite cases which are collected in the works of the authors at the head of my paper.

I have omitted a large number of cases which would only have been a repetition of the same fact, and also many others which, though interesting, were too complicated to be of service in determining what part of the nervous system was responsible for the skin lesion. Although I have not brought much new material together, I trust that a restatement of the facts with somewhat different grouping may be useful to those who are interested in the subject. I leave for others the discussion of the different theories that have been advanced to explain the relationship of the nervous system to the skin.

THE NERVOUS DISCHARGE.[1]

BY CHARLES MERCIER, M.B.

We eat and we work. These are the two great functions of the human organism—of all animate beings. All other functions, save the reproductive, are subservient to these two, and these two reciprocally subserve each other. We cannot work without eating; we work in order to eat; and we cannot eat unless we work.[2]

What happens when we eat? Food is taken into the body, and therein undergoes various transformations. It is dissolved, recombined, absorbed, circulated, combined into tissue, oxidised, decomposed, dissolved and ejected. We can sum up all these changes in one word—rearrangement. The atoms and molecules of the substances forming the food are rearranged into solutions, rearranged into blood-plasma, rearranged into tissue, and finally rearranged into combinations which are of no further service to the organism, and which are then expelled. The whole history of the food and the oxygen that are taken into the body consists in successive rearrangements of composition and changes, that is rearrangements, of place, from the time they enter the body until the moment they leave it. All the functions of mastication, deglutition, digestion, absorption, circulation, assimilation, nutrition, growth, secretion and excretion, are parts of a single process—the rearrangement of matter within the organism.

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[1] Not long ago an experienced alienist expressed to me in conversation his thorough disbelief in the existence of a nervous discharge. My surprise diminished when I found that the discharge of which he spoke was of the nature of a discharge of pus. Such a conception of the nervous discharge is so discrepant from that which is usual among neurologists, that I could not but look upon its existence as a reproach to Neurology. On search, I could find no statements of the nature of the nervous discharge, except the original exposition in Mr. Herbert Spencer's 'Principles of Biology and Psychology.' As these works are not, perhaps, universally in the hands of alienists, nor even of neurologists, I have thought it might be useful to set forth the nature of this discharge as now conceived by those who are most engaged upon the subject, in language as familiar and as free from technicality as the nature of the topic will allow. While following Mr. Spencer in the main, I have not scrupled to pursue the matter into further detail, and to make considerable additions and modifications which he might not approve. To me, therefore, belongs the responsibility for the views herein contained, while to Mr. Spencer is due the credit.

[2] The term "work" is used, not in its limited sense as equivalent to labour, but in the larger sense of producing movement. When I lift my hand to my mouth, there is work done which can be measured in foot-pounds.

How do we work? By moving our muscles. This is the one and only means by which we can alter the world outside of us. When a muscle contracts, a considerable movement of some part of the body occurs. But even when the muscle has repeatedly contracted, there does not result a large rearrangement of matter within the organism. A little of the muscular structure has been used up; a little new material has been introduced; but at the end of the exertion the arrangement of the muscular structure is but little different from the arrangement that it had at the beginning. Where then has all the motion come from, seeing that motion no more than matter can originate out of nothing? Obviously, the molar motion of the muscles and limbs has been supplied by the transformation of molecular motion. There has not been a large rearrangement of matter, but there has been a large redistribution of force within the organism. The redistribution of force in great quantities and to great distances, without any discernible rearrangement of matter, occurs in every electric-light lead and in every telegraph-wire. The contraction of the muscles is set up by the delivery of a shock of force at a nerve-end—another redistribution of force. This shock has travelled—has been redistributed—from a nerve centre. The nerve centre was provoked to emit its force by the arrival of a shock from some other region of the body, and we may continue to trace the communication of force to one part of the body from another, until we ultimately find that the whole process was set going by a force impressed on the organism by some moving thing in the outer world. At every step except the first, and in some cases the last, we find that the passage, or communication, or redistribution of force is the conspicuous, important and permanent part of the process; while the rearrangement of matter is trifling, or imperceptible, or temporary.

All the bodily organs and all the bodily functions may be divided into two great groups, according as they subserve the rearrangement of matter or the redistribution of force. The latter is, from the physical point of view, the prime function of the nervous system, of which the muscles, bones, &c., may be regarded as mere appendages.

Redistribution of force means, as we have just seen, the communication of force from one place to another. Matter is communicated from one part of the body to another in the blood-vessels, in which it is continually circulating. Force is communicated from one part of the body to another along the nerves, in which it also is continually circulating. Little waves of force pass along the nerve-fibres, at a rate of about 90 feet per second. The matter of the nerve does not appreciably change its place, the force alone travels, just as a wave passes through a fluid in the way familiar to every student of physics. But redistribution of force means something more than communication of force from place to place, and something more than this is included in the physical functions of the nervous system. Were communication of force the sole function carried on by the nervous system, we could never have a muscular contraction except upon the application of a proportionate stimulus to the surface of the body. The muscles are not always in full action. They contract when a shock is delivered into them by a nerve, and only then. It is obvious that we could not always depend upon having an amount of force, of precisely the requisite amount to cause a muscle to contract, applied to the body at precisely the moment at which it would be convenient for us to have such a contraction. Yet without such a stimulus applied just in the nick of time, no contraction could take place if the nervous system were solely an apparatus for the communication of force. In order that muscles should be made to contract at the right time, there must be a store of force accumulated somewhere in the body and capable of escaping from time to time in such quantity as to set up contractions in the muscles, without immediate and direct provocation from outside the body. To form such a store is the function of the grey matter. The function of the nerves is to form channels of communication along which force may be transmitted from one part of the body to another. The function of the grey matter is to act as a receptacle or reservoir for the storage of force, and to liberate this force at appropriate intervals, in appropriate quantities and in appropriate directions.

The conception of the storage of force is familiar to every one in the leading case of coal. Every one knows that this homely subtance derives all its value from the immense store of force it contains, and the ease with which this force can be liberated when we want it; and it is not difficult to form a moderately clear concept of the means by which the force is held in thrall. The classical illustration is the raising of a stone. When a stone is raised, a certain amount of force is used in raising it. If it is dropped, it strikes the ground with a force equal to that employed in lifting it. If it is again lifted and placed on a shelf, the force employed in lifting it becomes latent or potential. It is not manifested in any way, but it is still in existence, for however long the stone may remain on the shelf, if it is at last pushed over the edge it falls to the ground, and expends in doing so an amount of force equal to that employed in raising it years, or it may be centuries before. When the stone was placed on the shelf, the force employed in lifting it was rendered latent or placed in store; when the stone fell, the force was liberated. Now instead of the earth and the stone—one vast mass and one small one—imagine two very small particles, one of carbon and one of oxygen, and for gravitation substitute chemical attraction. So long as the carbon atom and the oxygen atom are kept apart, there is force potential or latent or stored up. When they clash together this force is liberated.

The method in which force is stored up in the grey matter is not, we suppose, quite like the case of the stone on the shelf, nor that of the restrained chemical action. Instead of a stone lifted, imagine a brick set up on end. To do this requires the expenditure of force. Now if the ground is shaken, the brick falls, and liberates in falling a force equal to that expended in raising it. Again, imagine a brick set on end with another brick placed across the top of it. The upper brick can now be knocked off the lower and the force which raised it be liberated, while the lower brick is left standing, with the force that raised it still in store. It is evident that a brick balanced on the top of another one will be displaced by a gentler shake than is required to knock down the single brick. A third brick may be placed on the top of the second, and this may be displaced by a still gentler shake, and its fall will liberate a still smaller part of the force employed in putting the three together. Now suppose more and more bricks are added, until we have quite a complicated structure composed of loose bricks. It is easy to see how readily a top brick could be knocked off—how slight a force would be necessary to upset it, how readily the fall of one would conduce to the fall of more, and how little the fall of a few of the topmost bricks would modify the entire structure, especially if we suppose that they do not fall off the pile, but merely from their ends on to their sides. Now if we imagine these bricks to be connected to the pile by elastic bases, so that when they have been knocked down they will slowly rise again, with perhaps a little help, to their erect position, we shall have a diagram which will represent very roughly what we suppose to be the mechanism of the nervous discharge.

We know that the grey matter of the nervous system has an extremely complex molecular condition. Each single molecule has been calculated to contain nearly one thousand atoms. These atoms are united together in twos and threes, in dozens and scores; several of these clusters are united together to form a larger group, which is again combined with others in several links of cross-relationship. The whole molecule is therefore a very complicated structure, and may contain many atoms and groups of atoms that stand in the position of top-bricks.

A structure so complicated is easily disarranged. When a disturbing force impinges against it, the weakest ties between the atoms and groups of atoms will give way, and some of the atoms will fall into new positions, just as the top bricks fall into new positions when the pile of bricks is shaken. Now mark, that in building up the atoms into the complex arrangement found in the molecule, force has been employed—has become latent —has been stored. And when the atoms are shaken into simpler positions, just so much force is liberated as would suffice to build them from this new position back into the old one. This is what is meant when it is said that force is stored up in the grey matter of the nervous system and liberated at intervals. Such a statement means, that the highly complex structure of the molecules of the grey matter is built up by the action of force on the atoms; that the force so employed lies latent in the structure; that the structure so formed is an unstable structure; that the bonds of union between the component sub-molecules are frail and easily broken; that the frailest portions of the structure may be shaken off by a very slight force, and that, when parts are thus shaken down, the force employed in building them up is set free. It is set free in much the same way as the force stored up in a pile of bricks is set free when the bricks fall down, and waves of sound and heat are straightway projected in all directions.

Such an alteration of the structure of a molecule as is above described, since it is an alteration from a more complex to a more simple structure, is of the nature of a decomposition, but since the molecule contains just as many atoms after receiving the shock and undergoing the rearrangement as it did before, there is no decomposition in the chemical sense. When a molecule is decomposed in the chemical sense, the atoms of which it is made up are not merely rearranged within the molecule; they are torn completely asunder, and may be united with foreign atoms to form entirely different compounds. This is not the case in the process that has been described. What happens there is a rearrangement of atoms within the molecule without any disruption of, or loss of atoms by, the molecule. The chemical composition of the grey matter is unimpaired, and a mere lifting of the displaced atoms into their original places will completely restore the *status quo ante.* Thus it can be easily understood how the structure of the grey matter may be continually undergoing decomposition during its functional activity without being permanently or materially impaired. It will be convenient to use the term "decompounding" for this form of rearrangement, reserving the term "decomposition" for the major process which involves destruction. The process of decompounding, with the liberation of force which necessarily accompanies it, together make up the nervous discharge.

Several accompaniments and consequences of this process require notice.

If we suppose, as the extreme complexity of the molecule and what is known of its constitution entitle us to suppose, that all the atoms and groups of atoms that occupy the position of top bricks are not equally unstable, but are bound to the main body of the molecule with different degrees of firmness, then it will be evident, that the number of atoms that are shaken out of their places on the impact of a shock will depend on the magnitude of the shock. A stronger shock will displace more atoms than a weaker shock; and since the greater the number of atoms displaced, the more force is liberated, it follows that, other things being equal, the *greater the disturbing force, the more powerful will be the resulting discharge.*

Next we have to notice, that the force set free by the decompounding of a molecule itself acts as a disturbing agent on the molecule. A very feeble impact may be able to displace only the most loosely attached atoms. But these atoms in their fall liberate force, and the force thus liberated may be enough to displace the atoms of the next degree of instability. The fall of those atoms may still further spread the process. Just as the fall of one brick on the top of a pile tends to knock down other bricks; just as the explosion of one barrel of gunpowder in a magazine tends to explode other barrels; so the fall of one atom of a molecule tends to knock other atoms out of their places. Hence *a discharge once begun tends to get stronger.* Again, the force set free by the decompounding of a molecule is not confined within the molecule, but diffuses around, much as the waves of sound and heat initiated by the fall of a pile of bricks spread around in all directions. But every molecule of grey matter is surrounded by other molecules. Hence the force set free from one molecule must impinge upon others. And when a force impinges upon the molecules of the grey matter, it will, if strong enough, shake their atoms out of place. Hence the discharge of a molecule tends to cause the discharge of other molecules. In other words, *a discharge once begun tends to spread.*

If a number of molecules are aggregated together, and some of them are made to discharge, a certain amount of force is liberated—the discharge attains a certain strength. From the two preceding paragraphs it appears that an increase of strength may accrue to this discharge in two ways. It may spread to more molecules, and be augmented by the force liberated from them, or it may spread deeper into each molecule and be augmented by the fall of additional atoms in each discharging molecule without any increase in the number of molecules discharging. The former is an extension of the discharge—an increase in the area that discharges with a given intensity; the latter is an increase in the intensity of the discharge that issues from a given area. Hence we recognise, that *a discharge may be augmented either by extending in area or by an increase of intensity.*

The force set free by the decompounding of a single molecule is of course inappreciably small: but the molecules are indefinitely numerous, and the accumulation of an indefinitely large number of inappreciably small forces of similar character results in a force of appreciable magnitude.

We have seen how the force stored up in the grey matter is liberated; we have now to ask how it is replaced. By what process, under the operation of what causes, do the displaced atoms return into their less stable position on the molecule? The only answer that we can at present give to these questions is that we do not know. We know that as a matter of fact the atoms are replaced, or that others are substituted for them, for the nerve-centres continue to discharge and yet do not become decomposed. It is obvious that, if the discharge continued without restoration of the fallen atoms, the process could not go on long; for each successive decompounding would reduce the atoms to simpler and simpler combinations, until at length the molecule would be reduced from a complex cluster of many groups of atoms and clusters of groups, to a disorderly heap of single particles. Long before this stage was reached, however, the molecule would split up into smaller molecules, and entirely new combinations would be formed. The grey matter would be chemically decomposed. We know, as a matter of fact, that no discharge is continuous. Every liberation of force is followed by an interval; and a prolonged discharge is made up of numerous separate waves of discharge separated by intervals of inactivity. It is fair to suppose, that during these intervals the fallen atoms are being replaced into a position of greater instability, from which they are again displaced as the discharge is renewed. This replacement of the fallen atoms requires time; it requires more time than is required for their displacement. Hence when discharge follows discharge rapidly and for a length of time, the process of displacement outstrips the process of restoration. The restoration of the fallen atoms being incomplete, the impinging force finds opposed to it a less number of top-bricks—of unstably placed atoms—in the molecule; and the topmost, or most unstably placed atoms being of course those whose restoration is the first to fail, the unstable atoms that are now exposed to the impinging force are less unstable than those which were at first exposed; they require a stronger impact to displace them. As the process continues, these atoms also fail to be replaced, and thus, as discharge is repeated, successive layers of atoms in the molecule are laid bare, each stratum being more stable than that which overlay it. At length a stage is reached at which the stability is too great to be overcome by the impact that is applied, and the discharge ceases. In this process we see the explanation of the occurrence of exhaustion and of the restorative potency of rest. After a long interval of rest so high a degree of instability is gained, that the impact of an infinitesimal force is enough to initiate a discharge, which then appears to be spontaneous, and which, once begun, is a cause of its own continuance. After a considerable discharge the degree of stability becomes such that stronger and stronger stimuli have to be applied in order to maintain the discharge. The stimulus of the voice, for instance, is replaced by that of the whip, and the whip is supplemented by the spur. On this hypothesis we see too why a short period of very vigorous exertion is more exhausting than much greater exertion spread over a longer proportionate time. In the latter case the intervals are occupied with the process of rebuilding, and their aggregate is sufficiently large to allow of a higher standard of structure being maintained than in the former.

By what agency this rebuilding of the molecule takes place we do not at present completely understand, but we know that there are many similar processes continually going on in the bodies of organisms. The development of the body out of scattered material by the accretion of molecule to molecule and cell to cell is a process of precisely the same character, and one of whose immediate causes we can give no account. The whole function of nutrition, of which the restoration to integrity of discharged nerve-centres is but a single instance, is a process of the same nature. All activity of function is attended by oxidation—by the degradation, that is to say, of some portion of the active tissue into a simpler molecular structure, and all growth, development and nutrition is the integration or redintegration of a simpler into a more complex molecular structure. Since the recompounding of the grey matter after discharge is a portion of the general process of nutrition, it will as a rule vary as general nutrition varies. Hence we find that those people who most readily recoup themselves after exertion—those who require least rest and least sleep—have the reputation of being the longest lived. Hence we find also that such people are the least obnoxious to adverse influences—to extremes of heat and cold, to the influence of alcohol and other drugs—and most readily overcome the effects not only of fatigue, but of various excesses.

The longer the molecules are left undisturbed, the longer does the building-up process continue, and the more and more unstable is the position in which their peripheral atoms become arranged. When a considerable time has elapsed since a molecule was discharged, the position of the atoms becomes so extremely unstable, that the impact of an infinitesimal force will be enough to upset them. It will further follow, that when atoms are in this extremely unstable condition, not only will they be upset by an extremely minute force, but that, once disturbed, the number of atoms that undergo rearrangement will be relatively great. Where a large number of atoms are in extremely unstable positions, a force which is sufficient to dislodge only the most unstable of all will, when it is reinforced by that liberated in the fall of these atoms, produce a widespread collapse; just as a house of cards falls into complete ruin when a single card is displaced. And the more atoms that are displaced, the more force is liberated, the more intense the discharge. Hence the longer a molecule has been left undisturbed, the more prone it is to discharge, and the more force does it liberate when the discharge occurs. In this elementary fact we see the explanation of the recuperation that takes place after rest, of the ease with which exertion of all kinds is undertaken in the morning, of the many phenomena of fatigue, exhaustion, &c. Here also is the explanation of the so-called spontaneous discharge, an occurrence which is never actually spontaneous, but which occurs when the molecules are fully charged, on the provocation of a force so small as to be insignificant. On the other hand, when the molecules have been frequently discharged and have had but few and short intervals for recuperation, the more unstable atoms will all be displaced; and as more and more stable strata are laid bare, greater and greater amounts of force are necessary to displace them; and when an atom is displaced, the connections of surrounding atoms will be so firm, that the displacement is limited to a small area, and the impact of even a considerable force will evoke but a very small discharge.

The function of the nervous system is, as we have seen, not only to store up and expend force, but to carry it from one part of the body to another—to deliver it from the store to whatever place it may happen to be wanted in. It remains to show how this transference is affected. It is scarcely necessary to say, that the liberated force exercises no choice as to the direction it shall take. It spreads from the point of liberation equally in all directions—provided all directions are equally permeable. But all directions never are equally permeable. Not only are the surrounding molecules, in so far as they are of the same nature, differently circumstanced, some being older, some more recently discharged, some better situated for recuperation, than others, but every molecule possesses polarity such that it is more sensitive to shocks arriving in the direction of one of its axes, than to shocks arriving in any other direction, and delivers its discharge in greater intensity along this axis than in any other direction. Hence a discharge will spread more readily where the molecules have their poles parallel than where they are higgledy-piggledy.

The more unstable the molecule, the more completely it is discharged by an impinging force, and the more force it liberates in its discharge. Again, the stronger force is the more efficient disturber, and hence for two reasons the discharge will spread most along the direction in which the molecules are most unstable. Now suppose that the unstable molecules are packed inside a tube whose walls are constructed of molecules so stable that they will not discharge at all, and suppose that they are packed with their polar axes parallel. Manifestly a discharge initiated at any point in the tube will travel with great facility along the length of the tube.

Such is the structure of a nerve-fibre. It is composed of an axis-cylinder of grey matter, enclosed in a tube of substance that will not discharge. We have seen that each molecule that discharges adds something to the force of the total discharge, and hence, if the supposed view is correct, the nerve current ought to be stronger the longer the tract of fibre that it has traversed; and it is a well-established fact in physiology that "if the irritant remains the same, the longer the portion of nerve irritated, the stronger is its action on the muscle."

Having such a construction as described, when a medullated nerve-fibre transmits a wave of force, this wave is entirely confined to the axis-cylinder in which it runs from end to end. The stably arranged sheath prevents all diffusion of the discharge. But all nerve-fibres do not possess a medullary sheath. Very many fibres are naked throughout their length, and all are destitute of sheath at their ends. From this we may infer that some nerves allow, under some circumstances, a certain lateral diffusion of the currents passing along them, and that this diffusion may occur at the ends of all nerves. We need not suppose, that where there is no sheath there is no restriction of this lateral diffusion, nor that where a nerve-fibre loses its sheath the currents spread from it uniformly in all directions; but we may be certain, from the existence of the sheath, that its absence allows to the currents some lateral diffusion. If not, there is no need for a sheath. When we look at a section of grey matter under the microscope, we see a number of fibres traversing in various directions an intervening substance. The fibres are not divided from the inter-fibrous substance by any sheaths or membranes. No conspicuous difference can be found between their substance and the substance in which they are imbedded. As a rule, the tissue must be considerably altered by immersion in different fluids, or by staining, or both, before any difference at all is discernible; and when the fibres are in this way made conspicuous, very many of them taper off to an extreme tenuity and become at length indistinguishable from the ground-substance. By more elaborate preparations, and by better methods of staining, the fibres may be tracked farther and farther; they may be traced into prolongations of greater and greater tenuity; but the more perfect our methods, and the more laborious our observations, the more certain it becomes, that the great majority of nerve-fibres taper off into fine points which lie imbedded in the ground-substance. From these observations three inferences may justifiably be drawn. From the different action of reagents upon the fibres and on the matrix, we may infer that the molecular constitution of the fibres is different from that of the matrix. From the precautions and preparations necessary to display this difference, we may infer that it is not very great. And from the increasing difficulty in tracing the fibres as they extend further from their main trunk, we may infer, that the difference between the molecular constitution of the fibres and that of the matrix diminishes as the former approach their free terminations. Viewing them with regard to their molecular stability, it can scarcely be doubted that the molecules of the fibres are more unstable than those of the matrix; but considering the similarity of the two substances, the difference of stability is probably not great, and becomes less as we approach the termination of the fibre. If we accept this view, in favour of which there are several arguments that cannot be reproduced here, we are committed to certain other views regarding the transmission of waves of force, or discharges along these fibres. Suppose a molecule in one of these fibres to discharge. Force is liberated, and, if the molecules of the fibre are supposed symmetrical and similar, spreads in all directions both along and across the fibre. When the discharge has spread through the whole thickness of the fibre, so that all the molecules in a transverse section are discharging, it impinges against the molecules of the surrounding matrix, which, by hypothesis, are not so easily discharged as the molecules of the fibre itself. It is obvious that the next event will depend entirely on the strength of the discharge. If the impact of the force is strong enough to knock down, not only the loose atoms that compose the molecules of the fibre, but also the somewhat more firmly compacted atoms that compose the molecules of the matrix, then the molecules of the latter that abut upon the fibre will be discharged. Discharging, they will liberate force, and will pass on the movement in increased intensity to the molecules lying further from the fibre. Hence, powerful discharges will not only traverse the fibre, but spread into matrix also. If, however, the discharge passing along the fibre has not sufficient force to upset the more stable molecules of the matrix, it will remain limited to the fibre, and will pass along its length without becoming laterally diffused. To suppose that the waves of discharge in the nerve-centres are entirely limited to the fibres, is a mistake, and a very vital mistake. Whether they remain confined to the fibres, or whether they overflow and permeate the imbedding substance, depends, other things being equal, on the strength of the discharge; and the possibility of their escape from the limits of the fibre into and through the surrounding substance is one of the most important properties of nervous tissue, and the foundation of one of the most valuable of all faculties—the faculty of progress.

Not only may a current, if sufficiently powerful, escape from the fibre and spread through the ground-substance, but there are circumstances in which a current in a fibre, however feeble, *must* become diffused into the surrounding matrix. Suppose the case of a fibre which tapers off and ends, after a longer or shorter course, as an attenuated point. What becomes of a current of force when it reaches this termination? Again, we must consider the process as an affair of decompounding molecules. If we take a number of cross-sections of the tapering nerve, the area of each section becomes less and less as we approach the point. The structure of the fibre being supposed uniform, it follows that the number of molecules exposed in each cross-section becomes less and less in each successive section. In other words, as the wave travels along the fibre, the discharge of a number of molecules is passed on to a less number. The force impinging on the less number is not less than the force impinging on the greater number. On the contrary, it is greater, for it is reinforced by the discharge of the latter. But the same amount of force when applied to a less number of molecules will produce a greater effect upon each than when applied to a greater number,—it will discharge the less number more completely. In other words, as the discharge approaches the termination of the fibre, it increases in intensity. When the discharge reaches the extreme point of the attenuated fibre it will have a considerably greater intensity than it had where the fibre was considerably thicker; that is to say, its impact against the molecules that lie round the point of the nerve will be more powerful than its impact against the molecules that bound the fibre laterally; and hence a discharge which is not powerful enough to escape laterally from the fibre, may yet have sufficient impetus to escape at its termination. When we remember that the molecules of the matrix, although more stable than those of the fibre, are not much more stable, it will be evident that when currents of various intensity are very frequently passing along the fibres, there must occasionally occur some that are not powerful enough to discharge those molecules of the matrix that surround the course of the fibre, but yet gather intensity enough to discharge those that surround its termination.

So far, we have discussed the nervous discharge without once mentioning the nerve-cells, an omission that may well arouse astonishment, but one that has been made designedly.

It is customary to regard the nerve-cells as unique bodies possessing wonderful and even miraculous powers. They are able, it is said, not only to transfer force from one fibre to another, but to transmute a wave of force into a conscious sensation, and an idea into a movement; they are able to suppress movements; not only force but memories are stored away in them; they are the active portion of the nervous system, the remainder being either mere packing or merely subsidiary and supplementary material. Most of these views are certainly erroneous, and all require modification. The nerve-cells cannot transmute a movement into a feeling or an idea into a movement. They cannot contain memories. They are not the only active portions of the grey matter. That they are active constituents of the grey matter admits of no doubt; but they are far from being the only or even the most important of such constituents.

The nerve-cells are continuous with the fibres. It is common to speak of a fibre as entering a cell; but the connection between them will be more correctly conceived if the cell is regarded as a bulging of the fibre, and the fibre as a prolongation of the cell. There are, it is true, cells which appear to be isolated, and which have no discernible connection with any fibre; and it is extremely significant that these cells have no cell-wall, and are less distinguishable from the matrix in which they are imbedded than any other form of cell; but the great majority of cells are continuous, with the substance of one or more fibres. Between the molecular structure of the cells and the molecular structure of the fibre there appears to be some difference, but assuredly the difference is not great. There is no line of division between them. The one merges by insensible degrees into the other. Their optical appearances and their response to reagents are almost identical, and whatever differences can be found to exist between them are largely explicable by the greater bulk and different form of the cell—by the different mode of aggregation of the molecules. Supposing, for the sake of argument, that the cells are composed of molecules precisely similar to those of the fibres, still the function of the cells, or the manner in which their molecules as a whole behave under the impact of a force, will be different from that of the fibre-molecules. Let us see what this difference will be.

The main difference in aggregation between the cell and the fibre, is that within any given distance, greater than the diameter of the fibre and less than that of the cell, there is a much greater number of molecules in the cell than in the fibre. Supposing a cell to have a diameter only ten times as great as that of a fibre, which is a moderate average, and to be of similar uniform molecular constitution, then, if discharges of equal intensity are started from the middle point in the long axis of each and spread to the circumference, the discharge in the cell will liberate 1000 times as much force as that in the fibre, and will occupy only 10 times as much time, so that in each unit of time the cell will liberate on the average 100 times as much force as the fibre. In other words, the discharge in the cell is of a more explosive character than that in the fibre, supposing both to be similarly constituted and to be similarly disturbed by equal forces. It is possible that the molecules of the cell are more unstably constituted than those of the fibre, but of this we have no evidence. But since most cells are a meeting-point for several fibres, it is obvious that while the impinging force which sets up a discharge at any point of a fibre can arrive in one of only two directions, that which discharges a cell may arrive from several directions, in some cells probably from any direction.

Since a large cell contains more molecules than a small one, it will, if similarly constituted, liberate more force in its discharge; and since the more explosive character of the discharge in the cell depends on the longer radius that can be drawn from the starting-point of the discharge to the periphery of the cell, it is obvious that a large cell will liberate force in a more explosive manner than a small one. Hence where an effect depends on both the amount and the suddenness of the discharge, the larger cell will have the advantage in both ways. It is significant that large nerve-cells are specially characteristic of motor regions.

Reference has been made to the fact, that some cells have no fibres in direct connection with them. Such cells—I speak only of those that are admitted to be nerve-cells—must have the same fundamental function as other nerve-cells—must accumulate force, and discharge it upon provocation. But since they have no fibres, the only possible way in which the jar necessary to upset the equilibrium of their molecules can reach them is through the matrix in which they are imbedded; and the only possible way in which the force liberated by the discharge of such cells can escape is through the matrix. Now we can see the significance of the fact already alluded to, that apolar cells are but indefinitely demarkated from the matrix in which they lie. Were it otherwise, they could exercise no function, for any boundary between the discharging molecules of the matrix and the charged molecules of the cell would prevent the former from acting on the latter. The same barrier would imprison the discharge and prevent it spreading. As it is, such cells may be looked on as but slightly differentiated portions of the matrix. They are separated from it by no defined boundary, and can freely receive and impart impulses from and to it. The existence of such cells is an additional argument for the active function which is here claimed for the matrix of the grey matter.

Having now considered separately in the fibre and in the cell the molecular movements which constitute their function from its physical aspect, let us see in what way these movements will be modified in their passage from the fibre to the cell and from the cell to the fibre. For the present purpose, those cells which are directly continuous with fibres may be regarded as bulgings in the course of fibres—as protuberances at a place of division. Now suppose a wave of discharge to proceed along a nerve-fibre and to arrive at one of these bulgings; what will happen? From the point at which the nerve joins the cell a discharge will spread through the whole cell-substance. The cell will discharge in an explosive manner, and will liberate a relatively large amount of force in a short time. This force, liberated by the molecules within the cell, seeks to escape. A head of pressure is established which tends to a restoration of equilibrium by flowing off in directions in which pressure is less. The force is pent up in the cell much as condensed gas is pent up in a soda-water bottle. It presses in all directions against its envelope. But the boundary of the cell is composed of material which is, metaphorically speaking, too rigid to yield to the pressure. The bounding molecules are too stable to be upset by any force that can be liberated within the cell. The force has to escape wherever there are openings in the boundary—in other words, wherever the cell-substance is prolonged into fibres; and these openings are few and small in comparison with the whole area of the cell. As in other cases where a considerable pressure escapes through few small openings, the rush through each opening will be of considerable intensity—of much greater intensity than would be the case if the force escaped freely over the surface of the discharging cell. We see, therefore, that for many reasons the discharge of a cell along the fibres emerging from it will be far more powerful and far more intense than the discharge whose entrance set up the discharge of the cell. In other words, a discharge in passing through a cell undergoes three changes. 1. Except in bipolar cells, which are few, it is communicated from a single fibre to several fibres. 2. It is increased in force. 3. It is increased in intensity. This increase in the force and intensity of the discharge is very great and sudden, and in comparison with it the increase gained by a discharge in passing along a fibre is insignificant in amount and, what is perhaps of more importance, is gradual in its accession. For these reasons the term "discharge" is often limited to the process as it occurs in the cell; the process as it occurs in the fibre, although identical in nature, being neglected.

So far we have dealt with the physical functions of the grey matter as occurring in a structure of constant composition. We have considered the force set free by the discharge of the molecules as travelling from place to place, becoming diffused, impinging against other molecules, and effecting their discharge. We have followed the course of the discharge from the fibre to the cell and from the cell to the fibre, and we have not forgotten that it may permeate the matrix also. But every disturbance that has yet been noticed is purely and necessarily a temporary one. If the change produced in a molecule by the rearrangement of atoms which constitutes its discharge were a permanent change, it is obvious that the molecule would be of no further use. Thenceforth it would be only an obstruction and an encumbrance. Only on condition that the fallen atoms are replaced, and the molecule restored to its previous condition, is a further exercise of function possible. All that we have at present considered is an oscillation of structure from a condition of less stability to a condition of greater stability and back again; this oscillation of structure being accompanied by a concomitant variation of function, the molecules now liberating, now accumulating force. We have now to notice that, besides this temporary effect, there is produced by each discharge a permanent effect, which is of enormous importance, and which is the basis of the psychological function of the grey matter, as the changes of which we have hitherto spoken are the basis of its physiological function.

Every wave of force that passes through the grey matter discharges all the molecules against which it breaks that are sufficiently unstable to be affected by it. Whether a molecule is discharged by a force, depends upon three conditions. First, on the constitution of the molecule as stable or unstable; second, on the degree to which, if unstable, it has recovered from a previous discharge; and third, on the direction with respect to its polar axis from which the force arrives. Some molecules, even of extreme complexity, such as those composing the medullary sheath of nerves, are of such stable constitution as to be incapable of discharge, and probably all molecules in the grey matter are not equally susceptible to this change. A molecule which has recently been deeply discharged and has not had time for recuperation, will not, it is manifest, suffer discharge except on the impact of a very powerful force. But supposing all the molecules of a tract of grey matter to have a similar constitution, and to be equally charged, then the facility with which a discharge passes through it will depend on the condition of the third factor—on the degree to which the direction of the wave approximates to the direction of the poles of the molecules. Suppose that the poles of all the molecules are parallel to one another, and suppose that the impinging force travels in the polar direction. Then it is obvious that the facility for the passage of the discharge is at a maximum. Such a molecular structure is the most permeable possible. But now suppose that the discharge passes through a structure in which the majority of the molecules have their poles in the direction of the course of the discharge, but some are placed obliquely at various degrees of inclination to this direction. Such a structure is manifestly less permeable to the discharge than the previous structure. Hence, when discharges of equal strength enter a given thickness of each structure, a less amount of discharge will emerge from the second tract than from the first. A residue of force equal to the difference between the two discharges on their exit has been retained in the second tract. What has become of this force? In what way has it been disposed of? *It has been used up in shifting the molecules so as to bring their poles nearer to parallelism.* Whenever a discharge passes through a tract of grey matter which has not reached its maximum of permeability, a part only of the discharge issues from the tract, and the remainder is spent in rendering the tract more permeable. The less permeable the tract, the greater the proportion of the discharge used up in increasing its permeability. The more permeable the tract becomes, the less the proportion of the discharge devoted to this purpose, and the greater the proportion that emerges. When the permeability reaches its maximum, the discharge that emerges is actually greater than that which enters, owing to the accession that it receives from the decompounding of the molecules. How the passage of a wave of force through an aggregate of molecules of different degrees of stability changes the less unstable molecules into more unstable ones, is perhaps not yet precisely ascertained. Those who desire to pursue the subject will find it discussed at length in Mr. Herbert Spencer's 'Principles of Biology,' §§ 3 to 23, and 304; and in his 'Principles of Psychology,' §§ 223 to 230. It will be enough here to quote two illustrations from those great works, which serve to give an accurate notion, not indeed of the kind of change that takes place in this particular instance, but of the manner in which a force in passing from particle to particle may be expended in two ways, part being absorbed in altering the disposition of the particles, and the rest being passed on to the extremity of the series of particles. "Take the rude analogy furnished by a row of bricks on end, which overthrow one another in succession. If such bricks on end have been adjusted so that their faces are all at right angles to the line of the series, the change will be propagated along them with the least hindrance; or, under certain conditions, with the greatest multiplication of the original impulse. For when so placed, the impact each brick gives to the next, being exactly in the line of the series, will be wholly effective; but when they are otherwise placed it will not. If the bricks stand with their faces variously askew, each in falling will have a motion more or less diverging from the line of the series; and hence only a part of its momentum will impel the next in the required direction. . . . Suppose that the row of bricks, which were at first very much out of parallelism, have fallen, and that part of the motion given by each to the next has gone towards bringing their faces nearer to parallelism; and suppose that, without further changing the positions of their bases, the bricks are severally restored to their vertical attitudes; then it will happen that if the serial overthrow of them is repeated, the actions, though the same as before in their kinds, will not be the same as before in their degrees. Each brick, falling as it now does more in the line of the series, will deliver more of its momentum to the next; and less momentum will be taken up in moving the next towards parallelism with its neighbours."

A further consequence of this increasing permeability of the tissue to the discharge will be the increasing limitation of the discharge to narrower and narrower channels. "Along a line of discharge, there is a genesis of the matter most capable of communicating the discharge. Every time an incipient nerve is traversed by another wave of molecular motion, there is apt to be a further formation of the molecules which are isomerically transformed by the wave and pass it on in being transformed. This process acts with continually increasing power, for two reasons. One is that progressing limitation of the wave to a well-marked line, enables it to produce more decided effects along that line. An illustration will here help us. When a body of water flows over a surface offering no distinct course, it thins out into wide-spread shallows near its margin, where it is almost motionless; and it has but little motion even along its central deepest parts. But if the inundation is long continued, the abrading action of the current along these central deepest parts where it moves fastest, tends to deepen its channel there more than elsewhere. A secondary result is a retreat of the water from the shallows—the current becomes more concentrated. In proportion as it becomes more concentrated, the force of its central part becomes greater still, and the deepening more rapid; which entails a further drawing-in of the margins and a further addition to the excavating force. So that the growing definiteness of the current brings a growing power of making its channel quite definite. Now though in the case before us we have not a motion of matter over matter, but a transfer of molecular motion from molecules to molecules, the parallel holds. Any greater effect produced by the transfer along one part of its originally broad course, similarly tends to concentrate the transfer along this part, and thus to intensify the action which makes this part a precisely marked channel. A further facilitation results from an absolute increase in the amount of the nervous discharge. The more permeable the line of molecules becomes, the greater becomes the initial quantity of molecular motion it draughts off. As with water, the formation of a definite channel not only makes the transfer easier and adds to the excavating power of the current, supposing its volume be constant, but also (if the reservoir can supply more) augments the volume carried away, which again adds to the excavating power; so the formation of a better line of nervous communication is followed by an increase of the wave that sets out to traverse it, and a consequent increase in the channel-making action. Once more, every addition to the molecular motion transmitted, adds to the effectiveness of each discharge in overcoming an obstacle. Suppose the greater part of its channel has become tolerably permeable, but that at some place in it the colloidal matter is less transformed than elsewhere into the fit type. Then the more the rest of its channel increases in permeability, the more powerful must be the wave of molecular motion brought to bear on the untransformed part, and the greater must be the tendency to transform, it. Hence the channel will progress towards a state of uniform permeability."

From what has gone before, the reader will have perceived that the freely permeable channels of communication, or the lines of aggregation, of the most unstable molecules are the nerve-fibres. This is common knowledge. What is not so generally recognised is that other channels of communication exist. Apart from the foregoing reasoning and from all question of the mode of origin of nerve-fibres, there is ample evidence of the existence of such other channels. It is a fact that a vast number—an overwhelming majority—of the nerve-fibres in the grey matter end at one extremity as fine points—have no discernible channel into which the waves of force that traverse them can be delivered. Each advance in our means of research enables us to trace these nerves a little further; but still their destination eludes us, the newest researches show us no more definite ending than a tapering point. If the matrix of the grey matter has no carrying function, each of these fibres is functionless. They are all histological abortions—tentative efforts, which have failed, to connect distant cells. An hypothesis which involves the supposition that the immense majority of nerve-fibres are so much waste material, is monstrous. According to the views that are here advocated, such fibres are merely portions of channels. They are those portions in which the process of mobilisation is complete; and they are supplemented by prolongations in which the process is incomplete—in which the molecules that have acquired the maximum of instability, and at the same time have acquired the power of acting differently on reagents and on light, do not constitute a sufficient proportion of the molecules forming the whole channel to give it, as a whole, optical and other qualities so distinct from the surrounding matrix as to enable us to recognise it as different. This is not mere hypothesis; for where we have a reagent which affects more powerfully than previous reagents these altered molecules, it enables us to trace the nerve-fibres further than a less powerful reagent. In other words, it brings into view prolongations of the fibres that were before invisible.

It is not, however, necessary to suppose that every nerve-fibre is prolonged by a definite, if invisible, channel to an ending in a nerve-cell. If the doctrines here advocated are correct, there must be many, very many fibres that have no such definite termination. It is one of the most vital principles of that neurology which forms the basis of psychology, that nerve-fibres and nerve-cells are not the only channels of communication in the grey matter. If the visible channels terminate in channels which are not sufficiently differentiated from the matrix to be visible, the inference is obvious that the discharge, which in passing along the visible channel is restricted to that channel, may, when it reaches the invisible channel, begin to spread into the matrix which is so little different. And if the fibres are gradually formed out of the matrix by the passage through it of discharges, then of course the matrix must be permeable to the discharge. This doctrine supplies functions at once to those apparently aimless fibres that, ending in free terminations, constitute so large a bulk of the grey matter, to the interfibrillar matrix which forms a still larger proportion of it, and to the apolar cells, which if visible fibres are the only channels of communication, are anomalous superfluities.

The conception of the physical condition of the grey matter that we have now reached is this. It is composed of complex molecules whose atomic constitution is such that they readily undergo internal changes without being disintegrated. These changes are of two main orders. One, which occurs on the impact of a force, consists in the fall of the atoms into more stable positions, and is accompanied by a liberation of force from the molecule. The other is the reverse change, and consists in the restoration of the fallen atoms to their previous positions, and is accompanied by a storage of force in the molecule. An illustration of these changes may be found in the waves of movement that pass over a cornfield under the pressure of the wind. Each stalk of wheat bends down under the impact of the wind into an inclined position, and in doing so strikes against other stalks and liberates the force which we recognise by rustling sound. No sooner is the bending complete, than the elasticity of the stalk asserts itself and the ear rises into the erect position, ready for the next gust of wind to bend it down again. The two movements occur in alternation, one under the influence of an external force, the other under the operation of intrinsic forces when the outside force has ceased to act. The greater the impinging force, the greater the depression of the ear. The more stalks that are affected, the wider the wave; the more each stalk is bent, the deeper the wave. Finally the wave of movement passes from one end of the field to the other without any redistribution of the stalks of corn.

All the molecules of the grey matter have not the same susceptibility to these changes, or, what is more probable, the molecules are not equally susceptible in all positions with regard to the disturbing force. If we imagine the stalks of corn to be oval in section, it will be evident that they will bend more readily to a wind in the direction of the short axis of the oval than to one in the long axis. If there be much difference between the axes, the stalks will not bend at all in the direction of the long axis; and if each gust of wind which strikes them on either side of the long axis is able to twist them slightly, so as to turn them more broadside on to a similarly directed gust, these properties will have a still more extended analogy to those of the molecules of the grey matter.

Such being the structure of the grey matter, we have now to get a clear conception of its function in physical terms. To do so, we must imagine the structure thus described as permeated continually and throughout by innumerable waves of discharge. We must recognise that there is a circulation of force in the body, just as there is a circulation of matter. The latter takes place mainly in the blood-vessels, the former mainly in the nerves; and the two are in many respects comparable. Along every nerve-fibre gushes of force continually succeed one another, as waves of blood pass through the arteries. Every nerve-cell is, as it were, a heart which receives the current flowing into it, and discharges it with increased impetus. Every tract of matrix is comparable with the intercapillary tissue. As the blood plasma soaks through such tissue in no constant direction, without definite boundary and with inconsiderable impetus, so the nervous discharge diffuses itself through the matrix of the grey matter in no definite channel, and with an impetus inferior to that which obtains in the nerves. As through the intercapillary tissue new capillaries are formed by protrusions from the old, so in the matrix of the grey matter new fibres are formed by protrusions from the old. Not only in the grey matter do these currents of force circulate; they are carried by the nerves to the uttermost parts of the body. There is scarcely a recess or nook of the organism to which they do not penetrate. While by far the greatest quantity is draughted into the muscles, large supplies go to all the tissues, where their access regulates those molecular disintegrations, rearrangements, and integrations that constitute nutrition and waste, secretion and excretion. The force thus expended is continually being renewed from the store that accumulates in the nerve-cells in the intervals of their activity. But however highly charged a nerve-cell may be, it never discharges spontaneously. However unstable its molecules, some force, even if infinitesimal, is needed to set the process going. Spontaneous movement, in the sense of movement originating without previous movement, is unknown—is, as far as we know the constitution of the universe, impossible. It implies a creation. We have to account, then, for the first shock that sets all the movement going. If we trace a discharge back from fibre to cell and from cell to fibre, we must always come at last to currents that arrive from outside the grey matter along the nerves that are called sensory or afferent. There is no other possible source for the first shake that sets all the movement going. And when we trace this afferent fibre to its extremity, we always find that it rises somewhere on the physiological surface of the organism—somewhere where forces from the outside world can act upon it, and originate the currents which, when they arrive at the grey matter, act as exploders. Further, we find that from all parts of the surface of the body nerve-fibres start and travel inwards to the great central masses of grey matter. And, furthermore, we find that several parts of the surface are especially modified, so as to collect and transmit to these nerves extremely slight disturbances arriving at the surface; such are the organs of special sense. To complete our conception of the physical function of the nervous system, we must therefore picture to ourselves waves of force starting from all parts of the surface of the organism, and converging towards the great central masses of grey matter. Here they are sparse and feeble, there they are stronger and more numerous; and from special areas, such as the retina and the internal ear, they run in powerful currents. Arriving at the grey matter, these currents join in and reinforce the general molecular turmoil which goes on there; and which eventuates in far more powerful currents that are continually distributed to the rest of the organism. The greater part of these outgoing currents are supplied to the muscles, and are supplied, as has been said, continually. So long as the muscles are at rest, the currents reach them in sufficient amount to maintain only that incipient contraction which we call their tone; but that even this is continually varying in amount is shown by the success of the muscle-readers, who can discriminate varying shades of muscular tension which are unknown to the individual in whom they occur. From time to time stronger draughts of force issuing from the grey matter reach the muscles in sufficient quantity to initiate the contractions by which our movements are made. Thus, partly as mechanical movement, partly as heat and other forms of molecular motion, the force accumulated in the nerve-cells passes out of the organism, and the redistribution, so far as the organism is concerned, is complete.

Clinical Cases.

ABSCESS IN CEREBELLUM: SYPHILITIC SYMPTOMS: SUDDEN BLINDNESS: GREAT OCCIPITAL PAIN: GREAT BENEFIT FROM CALOMEL: DEATH: AUTOPSY.

REPORTED BY MONTAGU HANDFIELD-JONES, M.B. (LOND.), M.R.C.P.

G. R., æt. 46, coachman, admitted to St. Mary's Hospital under the care of Dr. Handfield-Jones on December 27th, 1883, suffering with severe pain in the back of the head and neck, accompanied by retraction of the head.

Patient is a healthy-looking, well-nourished man; is married and has thirteen children, all healthy; his father died of heart disease, his mother of uterine hæmorrhage at age of 54, brothers and sisters alive and well. Was always strong and healthy till four years ago, when he was attacked with rheumatic fever, which confined him to his bed for four months. Following the rheumatic fever, patient was seized with paralysis of right side of body; this, however, passed away entirely, but two years ago an attack of total blindness, accompanied by mental aberration, came on suddenly; for this he was eight weeks in Maidstone Hospital. The total blindness lasted about two weeks, and then sight was gradually fully restored. Syphilis is positively denied, but there are two places on lower part of left leg which look like the scars of rupial sores, and higher up on tibia of same leg is a large node; over crest of the left ulna is another node. There is a scar on the centre of the sternum, which has existed for a long while; patient says he had a sore there which came by itself, and after some time discharged a small piece of bone.

Patient is very deaf, and has been so for many years; he talks apparently quite rationally, but his own account of his illness is not trustworthy. According to his wife's statement, he has suffered for at least four months with sickness and pain in the back of the head on rising from bed in the morning; during the last three or four weeks there has been severe pain in the back of the head and neck. The pain comes on in fits, which last two or three minutes, and are accompanied with retraction of the head. On December 24th, while at home, patient was very noisy and delirious; lately the attacks of pain have become much more frequent, recurring every half-hour or so. During the past week he has suffered with constant sickness; the bowels have not been open since December 19th, but he says he often goes for a fortnight without passing any motion, and for two days without voiding urine. There is no evidence of any accumulation in bowels or bladder; but patient has a stricture, which will not admit a No. 6 catheter. Heart, lungs, and other organs apparently quite healthy. Well-marked optic neuritis in both eyes; pulse 74, forcible, regular, not easily compressed; tongue coated, is protruded slightly to right; temp. 98·2; urine, straw colour, acid, sp. gr. 1035, no sugar or albumen, deposits crystals of oxalate of lime, contains some mucus and epithelium, total amount in twenty-four hours scanty. ℞. Calomel gr. v statim; leeches (12) to back of head; pot. iod. gr. x + pot. bromid. gr. xx + aq. ℥j, t. s. d.

Dec. 28th.—Patient passed a fair night, but the pain in the head was very bad at times; the attacks of pain occur rather less frequently. Vomiting took place after breakfast. Temp. this morning, 97·5. There was free bleeding from the leech-bites last night. ℞. Pil. hyd. c. col. gr. x statim; simple diet; beef jelly, O j; milk, O ij; ol. crotonis, ♏j + Ol. ricini, ℥ss.; ʒij to be taken at a time.

Dec. 29th.—Patient is fairly rational, complains of great pain in the back of the head, says he feels worse, but the attacks of pain are not nearly so frequent. The bowels have been open after two doses of croton oil and an enema. Pulse 84; temp. 97·5. Blister (3 × 3) to back of neck.

Jan. 3rd.—Had severe pain on night of 31st, but very little since; has been taking potass. iodid. in gr. xx doses, also calomel in gr. j doses three times a day since December 31st. Is sufficiently rational now to help himself to food, but passes his evacuations under him, and seems to have no recollection of it. Shows his tongue and does what he is told. Pulse 93, fair force; regular, constant, and seems to have power over his limbs; no paralysis. Repeat blister to back of neck.

Jan. 5th.—Is much more rational, passed a very good night, is able to open his locker and get at food he had left. An enema was given to-day with much ado, patient struggling violently and shouting murder, thieves, &c. A few hours later he protested that he had not shouted at all. Still passes all his evacuations in bed. Pulse 108, soft, regular, no ptyalism.

Jan. 8*th.*—Has more pain to-day in back of head and neck. Retraction of head more marked; evacuations passed in bed; patient less conscious. Leeches (12) to back of head; calomel gr. j, quater die; potass. iodid. gr. xxv + pot. bromid. gr. xxv + aq. camph. ℥j, quater die. Temp. last night, 97·8; this morning, 97·5. Pulse 80, full and forcible.

Jan. 10*th.*—Spasms of the neck are very marked; arching of the neck is very distinct. Patient not more conscious; was awake all night, but is dozing now. Rep. mist. c. potass. iodid. gr. xl. Calomel, gr. j, semel die.

Jan. 21*st.*—Patient has been passing quieter nights since last report, but is still very stupid and troublesome at times. The arching back of the head is still well marked. Pulse 58, interrupted by a pause about every eight or nine beats. Temperature this morning, 97. Since the 14th inst. he has taken ol. morrh. ʒj, semel die; yesterday he was ordered calomel gr. iij, ter die; pot. iodid. gr. vj + aq. ℥j, omni hora.

Jan. 25*th.*—Not much change since last report. Constipation has been troublesome, but has yielded to ½ ♏ doses of croton oil. At 4 A.M. this morning, patient was suddenly taken worse. Æther and ammonia were administered every quarter hour, and a sinapism applied over the heart. He has rallied and is now sleeping. The iodide mixture has been stopped.

Jan. 28*th.*—Patient much more rational. Gets up of his own accord to pass his motions. Recognises his wife and child. His wife notices how much more clear he seems in his head. ℞. Ammon. carb. gr. v + tinct. cinch. co. ʒj, + infus. cinchon. ℥j, quater die.

Feb. 1*st.*—Yesterday was suffering severe pain at back of head and neck, the latter was much arched back; pulse about 80, very irregular; was ordered calomel gr. ½ duabus horis. To-day seems quite free from pain, and the head is not drawn back. The bowels are open naturally and he gets up to evacuate. Patient has his spectacles on and is trying to read his newspaper, but does not seem to make much sense of it. Pulse 93, soft, regular.

Feb. 14*th.*—No pain whatever in head. Wishes to go out. Moves head freely in all directions. Gums touched by mercury. Reads newspaper fairly well, so as to be intelligible to bystander. Mental faculties quite clear now, memory returning. Left pupil noticed to be rather larger than right. Pil. calomel semel die.

Feb. 18*th.*—He has been steadily improving since the last report, and gets stronger every day. Says he feels better than at any time since admission; his memory has quite returned, and his mental faculties seem perfect. Gets up at 6 A.M., and remains up all day; he walks somewhat feebly, but there is no entangling of feet or anything specially noticeable in his gait. Pulse 88, good force, regular. Temp. last night 98·6, this morning 98·8. Has suffered since last report with an attack of diarrhœa, but the bowels are acting well now. Both eyes to ophthalmoscope show fundus blurred and hazy; no disc can be defined, and only a feeble, whitish, irregular patch; retinal trunks look large and hazy, small vessels not seen. He reads fairly well with glasses.

Feb. 22nd.—Patient has been failing since the 20th inst. Has complained of much frontal pain and suffered much with diarrhœa. Deafness rather increased. Undue fulness of frontal veins noticed. Yesterday it was observed that he seemed to have lost control over the sphincter ani, the bowels seem to act before he is aware of it. Last night he suffered much with pain at the back of the head; this has now passed off; the attacks of spasm and arching back of the head have returned, together with more or less of all the old bad symptoms. The diarrhœa is still troublesome, but only in the early part of the morning. Pulse 84, full, regular. Temp. last night, 98, this morning, 97·6. Urine passed involuntarily into bed during night. Has returned to the calomel gr. ½ ter die.

Feb. 23rd.—Much pain over the top of the head; headache more frontal now than at back. Pulse at 3 P.M., 63. Temp. 98. ℞. Calomel gr. j, duabus horis. Patient was seized with sickness at dinner-time; vomiting continued on and off till evening. Death at 7.20 P.M. apparently from cardiac failure.

Post-mortem examination on Feb. 25th.—On opening the skull, the convolutions on the convexity of the brain are seen to be much flattened; the sulci are obliterated, the superficial veins engorged. On attempting to remove the brain the right half of the cerebellum was found firmly adherent to the dura mater of the anterior portion of the posterior fossa, so that the dura mater was taken away with the cerebellum. Much fluid escaped from the rupture in the floor of the third ventricle produced during removal of the brain. The right lobe of the cerebellum, contained two abscess cavities lined by a pyogenic membrane, evidently not of very recent date. The cavities were close beneath the surface (rather nearer the upper than the under part) of the cerebellum, separated from each other by a septum ½ inch thick of tough brain-substance; both contained thick green pus. The dura mater corresponding to site of abscesses was adherent to the cerebellum over a space equal to the size of a crown piece, and was much thickened by tough, translucent-looking material, probably gummatous in origin and of the same nature as the morbid growth to be afterwards described on the periosteum of the tibia and ulna. The pia mater covering the cerebellum was thickened, especially near the site of the abscesses so probably blocking the foramen of Majendie and leading to distension of the ventricular system. In both eyes there was well-marked papillitis, though this was more apparent in right than in left; numerous small hæmorrhages were also well seen. The lungs, liver, spleen and kidneys were all intensely congested.

In connection with the periosteum of the left tibia there was a gumma equal in size to a walnut; on section, the centre was seen to be almost diffluent, while the outer firmer part was composed of grey, translucent tissue of a tough consistence. A gummatous growth of a similar nature was found in connection with the upper third of the posterior surface of the left ulna.

The morbid anatomy and pathology of the case recorded above are written in plain and distinct characters. In spite of his assertions to the contrary, it is clear that the patient had contracted syphilis at some period of his life; the scar on the sternum from which dead bone had been discharged, the gummata on ulna and tibia, the deposit in the dura mater over the site of the abscesses, are all so many definite and undeniable proofs. Again, it is obvious that the abscesses in the right lobe of the cerebellum were due to retrograde changes taking place in a mass of gummatous deposit, also that the distension of the ventricles causing flattening of the convolutions and obliteration of the sulci was caused by an accumulation of interventricular fluid, the escape of which was impeded by the pressure of the pia mater, thickened by inflammatory deposit, on the foramen of Majendie. Leaving, however, these clear and certain facts, we cannot fail to be struck by certain points in the history of the case, points which though of great interest in the clinical history, are yet by no means easy of interpretation:—thus, from what period in the history of the patient are we to date the onset of troubles due to specific cause? why with such a well-marked tumour were so many orthodox symptoms of cerebellar disease absent? can we fix the date of the onset of suppuration in the cerebellum, and can we account for the rapid return of all the bad symptoms, followed by death, when the patient seemed to all intents and purposes well out of the wood? The history given shows that the man was strong, able-bodied, the father of a large family, and in perfect health till four years ago; then comes an attack of rheumatic fever which confines him to his bed for four months, and following the rheumatic fever is an attack of right hemiplegia; now was this attack of rheumatic fever genuine and true, followed by a hemiplegia due maybe to cerebral embolism, or was syphilis once more proving itself the great imitator, and during those four months of mimicry laying down an intracranial neoplasm, which by its inhibitory effect on the brain functions should produce the right-sided paralysis recorded? The history of this period is unfortunately faulty, and we have no report of the progress of the disease or treatment adopted, nothing but the bare fact that he got to all appearances perfectly well, and remained so until two years ago. One point we may call attention to, viz. the heart was carefully examined while the patient remained in the hospital, and at no time was anything abnormal noticed in the sounds; this fact alone militates against the idea of a rheumatic fever of four months' duration followed by hemiplegia presumably of embolic origin.

The next stage in the history of the case dates back to two years ago, when an attack of sudden blindness, lasting two weeks, and accompanied by mental aberration (extending over two months), is noted. This loss of vision is interesting in connection with the papillitis, which was demonstrated during life and at the post-mortem; if Schweller's view as advocated by Dr. Hughlings-Jackson be accepted, it is highly probable that this early and brief loss of sight was due to a reflex inhibitory influence exerted by some fresh activity or change in the growth or relations of the cerebellar tumour. Such an explanation is in no way incompatible with the idea, first put forward by Manz, that dropsy of the intersheath space of the optic nerve was the cause of the optic neuritis recognised during the patient's last illness. The inhibitory influence exercised at the early period would be lost, as soon as the brain tissue adapted itself to its new relations with the tumour, or maybe as soon as certain active changes in the life of the tumour ceased, while the influence of the increased intracranial pressure, demonstrated post-mortem, would have a lasting influence on the nutrition of the optic nerves. The presence of mental aberration is probably to be accounted for in the same way by a temporary inhibitory influence brought to bear on the higher cerebral functions: that the action of specific treatment may have had an important bearing on the duration of the blindness and mental derangement is of course to be borne in mind.

Coming now to the third or last stage in the patient's clinical history, we find that during the four months preceding admission to the hospital the patient had been steadily going down hill. His wife reports, that on rising in the morning at 6 A.M. he would go down to the stables to groom his horses, clean his harness, &c., but after some half-hour's work would become so faint and dizzy, and have so much pain in his head, that he would be obliged to give in until after breakfast, when he would revive and be able to go out driving. By degrees this early morning faintness and pain in the head grew worse, and on rising from bed she had to give him strong beef-tea and a cup of strong coffee before he could start work; then gradually the pain in the head, especially at the back, became more continuous, till he experienced it all day long, and had at times to come home and lie down. The last three weeks before admission there was severe pain at the back of the head and neck, the pain coming on in fits, lasting two or three minutes, and accompanied by retraction of the head; towards the end of this time, i.e. shortly before his admission to the hospital, the higher cerebral functions were again attacked, and delirium with loss of memory is recorded. From this period till the date of death, the patient was under medical observation, and we have his symptoms accurately recorded. Some of these symptoms are such as we should clearly expect to be present, such as the constant vomiting, the pain in the head, the retraction of the neck, the constipation, the optic neuritis; but there are others which, in the light of post-mortem knowledge, we might equally have expected would not have been conspicuous by their absence. Thus at no time do we find the ataxic walk, the feet becoming entangled one with the other; no irregular movements of the eyeballs are recorded; there is no observation made as to the existence of tonic convulsions. Regarding the two latter symptoms, there does not seem to be any good recognised reason why they should be present in some cases of cerebellar disease and absent in others; but as regards the ataxic walk, I find that in a series of fourteen cases, notes of which I have by me, in only two instances was this symptom present, and in both these patients the mischief was very extensive and involved the central parts of the organ. With respect to the abscess in the cerebellum, the condition of the pus, thick, green and curdy, showed plainly that suppuration had occurred some considerable time previously; and as the temperature during his stay in the hospital was throughout subnormal, and none of the usual signs of suppuration setting in ever manifested themselves, it is highly probable that the purulent degeneration had taken place previous to admission. The extremely diffluent condition of the central part of the gumma on the tibia showed how ready the new tissue was to degenerate into pus; and granted a similar condition in the central part of the brain deposit, it is quite possible that the degenerative condition may have gone on so gradually that severe constitutional disturbance was at no time excited. Nothing could have been more marked than the steady improvement which took place from February 1st to the 20th of the same month, and it may reasonably be suggested that the exhibition of small but continued doses of calomel played an important part in bringing about so very marked a gain, especially as all the bad symptoms returned on the drug being gradually dropped. It is plain, however, that the attack of cardiac syncope on the afternoon of January 25th showed on what precarious tenure life was held, and it was probably to some similar inhibitory influence brought to bear on the heart, that the death of the patient on February 23rd was finally due.

A CASE OF IDIOCY WITH UNIVERSAL RIGIDITY, THE RESULT OF SYPHILITIC DISEASE OF THE CENTRAL NERVOUS SYSTEM.

BY ANGEL MONEY, M.D., M.R.C.P.,

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A. R. H. a female child, aged 3½ years, was admitted into the Hospital for Sick Children, under the care of Dr. Dickinson, on April 3, 1883. She was an illegitimate child; her mother died of consumption, aged 31. The child had been bottle-fed all through. She was in good health till two years old, when she had two "fits" (? bilateral) in one afternoon, which were attributed to the teething. Three days after these fits, the child is said to have become unconscious. Since then she had not recognised friends, had not spoken, nor walked; though before the fits she was intelligent, could speak and had walked well. The bowels had been constipated. There was no vomiting.

On admission, the gums were spongy and swollen; the bowels were freely opened after a purgative; the patient vomited three times without apparent cause; the temperature was 104 at night, and 101 in the morning; the corneæ were clear; the discoloured teeth were ground down level with the gums in the upper jaw, and projected a little beyond the gums in the lower jaw. The upper part of the right external ear stood away from the side of the head. Unless the optic discs were too pale, ophthalmoscopic examination revealed nothing abnormal. The eyeballs were moved about thoroughly in every direction; they had no characteristic position; there was no facial palsy in crying; the tongue seemed to move naturally and equally. There was practically no intellect. The child seemed to observe a bright lamp, cried when food was put into the mouth; deglutition was natural. The mouth was very large, the face seemed ill-developed; the head was brachycephalic and small; its circumference measured 18½ in.; the measurement from ear to ear over the cranial vault was 12½ in. The legs, arms and trunk, when left alone and at rest, were not stiff, or but very little so; the slightest disturbance however (sometimes the same thing seemed to occur apparently spontaneously) developed a marked tonic spasmodic condition of all the limbs, with opisthotonos of all parts of the spine, cervical, dorsal, but especially lumbar. The legs were straight at all the joints. The arms were rotated in and adducted at the shoulder; were extended at the elbow, but not fully; pronated in the forearm; the thumb was turned into the palm and the fingers flexed thereon, the wrists were semiflexed. This posture of parts was constant, fixation occurring when spasm came on; the onset did not recall a spasm the result of strychnia poisoning or tetanus, principally in the want of suddenness, but perhaps there might be less difference between them than was supposed. The knee-phenomenon could be got on each side without difficulty; the ankle clonus could not be obtained, on account of the rigidity.

April 13th.—The patient was in much the same state, but the gums were practically well. The temperature had been below the normal since April 8th.

April 16th.—The patient swallowed badly, and sometimes did not attempt to swallow at all. The food regurgitated through the nose. Vomiting had been frequent.

April 17th.—The patient swallowed better in the night, but this morning she was worse again, and a muco-purulent discharge was flowing from the mouth constantly; there was no cough nor vomiting; the muco-pus bubbled up in the mouth. The bowels were constipated; a nutrient enema was given, which returned in half an hour. The voice became very weak last evening, so that she had not screamed when disturbed; this was a new feature. The back and neck were much more arched.

April 18th.—Excessive pallor appeared at 6 A.M., and was universal and ghastly. There was marked collapse. The patient was pulseless, the surface cold, the breathing shallow and actually ceased at times; the lips were bluish. The rigidity had more or less disappeared when the patient was disturbed. The hands were clenched, but could be undone; the fingers were flexed, the thumbs turned, the arms adducted, the elbows gently flexed, the forearms over-pronated; the legs were extended at all the joints; there was conjugate deviation of both eyes to the right. Death took place at 7 P.M.

Autopsy.—The weight of the body was 17½ lbs. The limbs were stiffened in the posture described during life. The cranium was asymmetrical, the left fronto-temporal region sloping, and not so round and full as the right. The frontals and parietals were 7 mm. thick at the thickest parts; the diploë was dense; the fontanelles were closed, and the sutures were united, but there were no bosses. The dura mater was decidedly thickened, especially about the walls of the sinuses; there was nothing abnormal in the venous sinuses; there was much œdema of the subarachnoid space; irregular opacity and thickening of the pia mater were observed chiefly over the sulci; the pacchionian bodies were fairly marked; the brain was atrophied, especially its left hemisphere; its walls in the lateral and posterior parts were quite thin; the convolutions for the most part were picked out and well defined; many of them had a yellowish hue and were quite tough. The walls of the basilar artery were opaque white, thickened, and contained red tough thrombus. Nearly all the pia-matral vessels were thrombosed and their walls diseased. Nearly all the large arteries had thickened and opaque walls; long lengths of vessels were thus diseased; the whole of the basilar and the terminations of the vertebrals were very conspicuously diseased, and by far the greatest part of the length of the vessels forming the circle of Willis was diseased. The orbital plates crushed rather than fractured with the chisel, their diploë was thick and vascular. There was nothing abnormal in the choroids or optic discs. The orbital fat on both sides was dotted with points of hæmorrhage. The middle ears contained muco-pus, but the membranes were not perforated.

The pons Varolii was very hard. The cerebellum was perhaps a little tougher than natural; each half was equal, and of about the same size and shape. The medulla and part of the cervical cord stand out like a stick, before being put into spirit. All the spinal cord felt hard; on section, the anterior horns in places looked puckered and indented.

The heart weighed 2 oz., was healthy, as was also the pericardium. The larynx had a slight shaggy appearance, and red specks were dotted about, but nothing more definite was seen.

The left lung was adherent nearly all over, its weight was 3¾ oz. An area of consolidation, of ill-defined outline, of a dark dirty-red colour mottled with white, and of diminished consistence, was situated in the middle of the left upper lobe.

The right lung weighed 4¼ oz.; the upper posterior part of the right lower lobe, and the back half of the upper lobe were consolidated, of dirty dark-red colour, mottled with white, of ill-defined outline, and of diminished consistence. The bronchial tubes were all dilated, in one place having the finger-glove look; there was much mucus in the tubes. In the large intestines only the first six inches showed the lymphoid structures to be of a yellow tint, of a circular or oval well-defined outline, with a central dimple. None measured more than 3½ millimetres across, many were smaller.

The liver weighed 12 oz., was of a dark red colour, and of diminished consistence; there were no gummata. The pancreas was healthy. The spleen was big, of a dark red colour, weighed 2 oz., its consistence was not increased. The bronchial glands were enlarged; there were no cheesy glands anywhere. The kidneys weighed 2½ oz. together. The bladder was healthy. The muscles appeared to be a little pale.

Microscopical Examination.—Sections of the spinal cord show a diffuse form of sclerosis throughout the whole of its length. The cerebral substance has not been microscopically examined, but it may be assumed that extensive sclerosis is present in those convolutions which felt hard at the autopsy. Many sections made from the basilar and middle cerebral arteries exhibit well-marked arteritis, chiefly of the inner coat; in some sections the chief thickening is between the corrugate elastic lamina and the outer coat. Complete microscopical examination has not been made, owing to the fact that the specimen was kept in spirit to preserve its naked-eye appearances, and the spirit has now so altered the texture of the brain and cord as to render a complete examination impossible.

Remarks.—The case is of interest, as an example of the production of idiocy by coarse syphilitic lesions of the brain. In my own experience, which is limited to four post-mortem examinations of children idiotic from or soon after birth, some gross organic lesion has been present, and was in all probability the cause of the amentia. In one case of microcephalus I assisted at the autopsy with Dr. T. Barlow, and the cerebral hemispheres were, roughly speaking, converted into thin walled cysts over the greater part of their extent. In my second case there was a tumour, apparently gummatous, the size of a Tanjerine orange in the posterior region of the right hemisphere. The third example was one in which there was great atrophy of the right hemisphere, which was probably due to hæmorrhage, the result of instrumental delivery.[1] The last instance is the one here recorded. All the specimens are in the museum of the Hospital for Sick Children, Great Ormond Street.

My clinical experience at the hospital last mentioned leads me to believe that mental changes are by no means uncommonly produced by hereditary syphilis. I have met with children in whom the evidences of past syphilis were unmistakable, and in whom the mental condition was obviously not up to the average. This subject has been drawn attention to by an excellent contribution to 'BRAIN,' from the hand of Dr. Judson Sykes Bury, of Manchester.

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[1] 'Transactions of Clinical Society,' 1883.

Reviews and Notices of Books.

A Treatise on the Chemical Constitution of the Brain.
By J. L. Thudichum, M.D.

This work treats, in the first instance, of brain matter as a whole; several of its constituents are next studied, and an attempt is then made to obtain an insight into its normal and abnormal chemical functions.

The abundance of water in the neuroplasm is the first striking fact that meets the inquirer, and this, in conjunction with the peculiar manner in which the water is combined, engenders a great mobility of the ultimate particles.

The brain contains a considerable amount of an albuminous base. The bearing of this albumen, the author states, is governed by the components peculiar to the brain, which also by their manifold chemical affinities assist prominently in producing its singular mechanical arrangements, its chemical function, and its sensory and volitional action and reaction. These components he arranges in three groups: the members of the first contain five or six elements, one of which is phosphorus, and they are therefore termed *phosphorised bodies;* the members of the second group contain four elements, amongst them nitrogen, but no phosphorus, and are therefore termed *nitrogenised bodies;* while the members of the third group contain only carbon, hydrogen, and oxygen, and may be termed *oxygenised bodies.*

The phosphorised bodies the author names *phosphatides*, on account of their similarity to ordinary phosphates, and of these he gives several subgroups and species, of which lecithins, kephalins, and myelins are examples. These phosphatides Dr. Thudichum considers to be the "centre, life, and chemical soul of all bioplasm whatsoever, that of plants as well as animals;" and amongst their physical properties none, he thinks, are more deserving of further inquiry than those he describes as their powers of *colloidation,* without which "no brain as an organ would be possible, as indeed the existence of all bioplasm is dependent on the colloid state." When this hydric colloidation is at a maximum, the tendencies to decomposition seem to be at a minimum. These phosphorised bodies are readily able to combine with acids, alkalies or salts: but all their affinities, whether acid, alkaline, or alkaloidal, are overcome by water in quantity; the affinities for water, however, are overcome in turn by some metallic oxides, such as those of lead, copper, manganese, and iron. The diversity of affinities is therefore very great, their exercise being greatly influenced by the mass of reagent and the mass of water present, and their interchange, accordingly, may produce an incalculable number of states of the phosphorised principles, and consequently of brain matter. From which it follows that neuroplasm, so far at least as the phosphorised principles are concerned, must yield obedience to even the slightest external chemical influence reaching it by way of the blood, taking up metals, acids, alkalies, salts, and alkaloids when so presented.

As to the colloid state above referred to, the author believes that its destruction in myoplasm, produced by the direct influence of the disease-germ, leads to the fall of temperature in the collapse stage of cholera. Some bacteria possess the power of liquefying colloids, and the phosphatides may be capable of being similarly affected. The so-called softening of the brain consists, in the first place, merely in the loss of the colloid state by a smaller or larger portion of the nerve marrow. Such a softening may be effected by the bacilli of tubercle. After fluidification comes patholysis, the splitting up of the liberated immediate principles into their proximate nuclei. The two processes as seen, one in cholera and the other in softening of the brain, illustrate the acute and chronic forms of loss of colloidation and of patholysis under the influence of microzymes.

The *nitrogenised non-phosphorised* substances of the brain imitate in some respects the properties of the phosphorised, but as compared with them, their chemical characteristic is slight solubility; they are all firm compounds, and do not easily oxidise or decompose. Six groups are included under this head, of which phrenosin, psychosin, kerasin, and bregenin are examples.

The group of *oxygenised non-nitrogenised* principles consists mainly of alcohols and organic acids.

The brain, on the whole, is an aggregated mass of bioplasm, deriving its peculiarity mainly from specific chemical additions. The stroma of this bioplasm is made up of albuminous substances. Among these are small quantities of soluble albumen, partly exhibiting the properties of serum albumin; small quanties of fibrin, and considerable quantities of a globulin-like body, to which the author applies the term neuroplastin.

Into the methods of research pursued by Dr. Thudichum we shall not enter, further than to mention that by means of them he separates the brain into what he terms:

1. *First extractives.*
2. *Insoluble albuminous residue* containing plastin, albumen, and collagen, &c.
3. *White matter* obtained from alcoholic extracts, containing kephalins, myelins, cerebrins, cholesterin, &c.
4. *Buttery matter* by partial concentration and cooling of the alcoholic filtrate from the white matter, containing kephaloidin, paramyelin, more lecithin, &c.
5. *Last oily*, obtained by further distillation of the alcoholic

filtrate from the buttery matter, and containing lecithin and paramyelin, &c.

6. *Ultimate watery extracts* containing alkaloids, salts, &c.

To the mode of separation, the reactions, and the formulæ of many of the phosphatides the author devotes a large portion of his book. The constitutional formula, for example, of the principle kephalin is given as

$$C_3H_5\left\{\begin{matrix}C_{17}H_{28}O_3 \text{ (Kephalyl)}\\ C_{18}H_{35}O_2 \text{ (Stearyl}\\ HO(OPO)\ C_5H_{12}NO\end{matrix}\right\} = C_{43}H_{81}NPO_8$$

The conclusions to be drawn from the chemolyses of these phosphatides are thus stated. No glycero-phosphoric acid was obtained in any of them, and therefore glycerol cannot be essential to the composition of these bodies. Their only common feature was the presence of phosphorus, which by ordinary analytical processes was removed in the form of phosphoric acid; but there was no reason to suppose that the phosphorus was present in the organic principles in any other form than phosphoryl (PO).

The quantitative relations of the immediate principles and constituents of the brain are fully discussed from Dr. Thudichum's own standpoint, and synopses are given of analyses of the grey and white substance of the human brain. The mean sp. gravity of grey tissue is stated to be 1·032; of white, 1·041; and of the entire brain, 1·037;—numbers which differ slightly, or not at all, from those commonly received. Of white tissue he assumes there is present 55 per cent., and of grey 45 per cent. in the entire brain. A useful sketch of a systematic quantitative analysis of the brain is next laid down, and this is followed by a table showing the result of quantitative analyses of the human brain. From the latter we shall make a few selections. The quantities given, it should be said, claim to be minima only, while the losses registered are confessedly great.

HUMAN BRAIN.

——	Grey tissue.	White tissue.
Water expelled at 95°	85·270	70·230
Neuroplastin	7·608	8·630
Kephalin, lecithin, myelin, &c. .	2·730	11·497
Cerebrins and myelin	0·434	6·910
Inosit	0·193	0·215
Lactic acid	0·102	0·045
Phosphoric acid	0·017	. .

"When the normal composition of the brain," as the author observes, "shall be known to the uttermost item, then pathology can begin its search for abnormal compounds or derangement of quantities. Thus the amyloid degeneration is specific to brain and nerve tissue, and can be considered hypothetically as a reduction of an ingredient of decomposed cerebrosides." The great diseases of the brain and spine, such as general paralysis, acute and chronic mania, melancholia and others, the author believes, will all be shown to be connected with special chemical changes in neuroplasm. The knowledge of the composition and properties of this tissue and of its constituents will materially aid us in devising modes of radical treatment in cases in which at present only tentative symptomatic measures are taken. Indeed, there is great probability in his assumption that, by the aid of chemistry, many derangements of the brain and mind, at present obscure, will become accurately definable, and more amenable to precise treatment.

The opportunity may here be taken of referring to a paper, published by Dr. Thudichum a short time ago, on the chemical diseases of the brain and spinal cord. He here assumes, that from the chemical constitution of an organ we may deduce the pathological changes which must necessarily take place in it under certain conditions, particularly after the ingestion of drugs possessing special affinities for some of its constituents; and that treatment must be based upon a knowledge of these affinities. Locomotor ataxy, chronic alcoholism, lead and arsenic poisoning, are adduced as examples of these chemical diseases; and in connection with the same, some of his remarks on what he designates cerebrose diabetes, a kind of acute glycosuria, are highly interesting. But into these, or his criticisms as to amyloid degeneration, we cannot here enter, nor are we disposed to agree with his opinions as to the constitution of amyloid substance.

Dr. Thudichum thinks it highly probable that phosphorus, an ingredient present in the brain in such a small proportion, may yet be its most indispensable constituent. Phosphorised substances are found in every centre of vital activity, and seeing that they can combine, in the state in which they exist in the nervous system, with almost all the chemical reagents with which they are brought in contact, provided the latter are in sufficient relative excess, these combinations in turn readily breaking down again, it is highly probable that they are closely concerned with much of brain disease. Lead-poisoning, he observes, being regarded as nerve poisoning, the kephalin and myelin combining with this body, the object of treatment must be to break up these combinations by the administration of a body capable of removing the lead from its combinations. The combination with myelin is very stable, that with kephalin quite unstable. Both these bodies have acid characteristics, the myelin behaving as a dibasic acid; while in apomyelin and lecithin the alkaloidal character prevails, and accordingly they combine more readily with such reagents as cadmic or platinic chlorides. Now, in the case of chronic lead-poisoning, potassic iodide has long been recognised as an efficient curative agent, and it is further known that it acts more readily when large quantities of water are administered with it. When a solution of this salt is brought in contact with a solution of a lead salt, iodide of lead is at once formed and potassium set free; and this is what occurs when it is given in cases of lead-poisoning. The water administered now plays its part by deluging the vessels with a solution of potash, which is not only a highly diffusible liquid, but also a stimulant of liquid secretion. The lead iodide is soluble in the potash solution, and as fresh quantities of potassic iodide are being constantly added, the lead combinations are more and more broken down, and thus the system is gradually freed from its presence.

Many kinds of headache, according to the author, are probably due to intracranially brewed chemical poisons, or to poisons carried to the brain by the blood from the body, having been fermented there, or, like alcohol, morphia or fousel oil, formed outside the body. "Many forms of insanity are unquestionably the external manifestations of the effects upon the brain substance of poisons fermented within the body. These poisons we shall, I have no doubt, he able to isolate after we know the normal chemistry to its uttermost detail. Then will come, in their turn, the crowning discoveries to which all our efforts must ultimately be directed, namely, the discovery of the antidotes to the poisons, and to the fermenting causes and processes which produce them."

Very much of the chemistry of the brain has, in our opinion, been omitted from the author's present treatise. He has neglected almost all reference to the work of anybody but himself. With much self-satisfaction, however, he thus excuses himself: "The literary discussions on subjects of brain chemistry which have taken place during the last few years, and in which I was obliged to take an active part, have had such an issue that I was happily enabled to exclude all controversy from the pages of my treatise." Accordingly, while we are disposed to regard Dr. Thudichum's treatise as a valuable contribution to the chemistry of the brain, we certainly cannot look upon it as embodying what is known as to its chemical constitution: and time alone will tell whether or not his contributions will be received as part of the great foundations upon which future researches may be safely superimposed.

T. Cranstoun Charles, M.D.

Mr. Sydney Hodges *on Retinal Images*.—I. "*After-Images*." 'The Nineteenth Century,' October, 1883. II. "*On some Peculiarities connected with Retinal Images*." 'Brain,' April, 1884.

The repetition by accurate observers of experiments previously and even repeatedly made by others is seldom altogether without value. When therefore Mr. Hodges has been led by an accidental occurrence to make a careful and able study of the after-images resulting from the fixation of very luminous objects, it might seem unnecessary to point out the large amount of work already done covering the same ground. Were it not indeed that Mr. Hodges expressly urges the prosecution of investigations such as he has undertaken, as likely to be productive of novel and interesting results, we need hardly have referred to this subject in the pages of ‘BRAIN.’ But it is important to remember that such experiments are not unattended with danger. Two eminent physicists, Plateau and Fechner, had occasion to regret the ardour with which they prosecuted researches of this nature. The former, with the view of deterring others from running the same risk, which cost him so dearly, not only repeatedly gave warning of the danger, but took the trouble to make a very complete bibliography of this subject.[1] A reference to that bibliography will convince any one that the main facts with regard to the effects produced by experiments of the nature of those under consideration, as far as they give rise to sensational changes, are well established. There is so close an agreement in the chief results described by most observers, that no further confirmation is required. So far as any theory or explanation of the colour-sense is concerned, these are available, and must undoubtedly be taken into consideration. Almost all modern physiologists who have occupied themselves much with the subject of visual sensations, such as Helmholtz, Hering, Brücke, Exner, Fick, Aubert, and a host of others have given more or less attention to this subject. Some, indeed, have advanced it far beyond the stage to which Mr. Hodges has arrived in his researches, and in ways which to explain would hardly be suitable matter for the pages of a neurological journal, but which those interested in the theories of vision will find to throw much more light on their speculations. It is true that Helmholtz in his ‘Physiological Optics’ does not devote much space to Mr. Hodges' subject, but he will find in it at least one very definite statement which I quote from the French edition, § 23. He there remarks: ‘Les objets colorés ne sont pas les seuls qui donnent des images accidentelles colorées, les objets blancs en fournissent aussi, qui présentent ordinairement des modifications de couleurs très variées. Nous donnons à ces variations le nom de phases colorées des images accidentelles. La succession de couleurs diffère avec la durée et l'intensité de la lumière première.”

The phenomena seem to have been first correctly described by Porta in 1593, though observed by Aristotle and Themistius, the

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[1] Plateau: “Bibliographie analytique des principaux phénomènes subjectifs de la vision depuis les temps anciens jusqu'à la fin du 18me siècle, suivie d'une bibliographie simple pour la partie écoulée du siècle actuel.’—*Troisième Section.* “Images qui succèdent à la contemplation d'objets d'un grand éclat, ou même d'objets blancs bien éclairés.” (In which the writings of twenty-six authors on this subject up to the present century are analysed, and fifty-two others of later date cited.) This section, as well as the complete bibliography, is prefaced by the following warning: “Les experiences qui font l'objet de cette troisième section sont dangereuses; c'est à la suite d'une expérience imprudente de ce genre, que s'est développé chez moi le germe de l'affection qui a fini par me priver complètement de la vue. Je ne saurais donc engager trop fortement les physiciens et les physiologistes à s'abstenir de semblables essais, qui ne présentent qu'une importance bien minime à côté des maux qu'ils peuvent entraîner. D'ailleurs, les faits observés jusqu'ici sur cette matière sont, je le pense, assez nombreux pour qu'on puisse se dispenser d'en recueillir de nouveaux.”

Abstracts of British and Foreign Journals.

Schultze on Secondary Degenerations in the Spinal Cord. —The author (*Archiv f. Psych.* Bd. xiv. p. 359) bases his paper on the following five cases:—(1) Compression of the cauda equina; (2) crushing of the lower half of the lumbar enlargement and the sciatic portion of the cauda equina; (3) atrophy and degeneration of the greater part of the lumbar enlargement, the result of fracture of the 1st lumbar vertebra; (4) breach of continuity in the cord at the level of the 9th dorsal vertebra; (5) perimeningeal tumour in the cervical region with atrophy of the middle third of the cervical enlargement from pressure.

First, as regards degeneration in the posterior columns. Schultze finds that immediately above the seat of injury the posterior columns degenerate in their entirety; further upwards the degeneration is confined to the inner portions of the columns, in an especial manner to Goll's columns. This rapid diminution in the extent of the degeneration is due to the fact that two kinds of fibres are represented in the posterior columns; a short set of fibres that spring from the posterior spinal roots and soon end, probably in the cells of the posterior cornua; and a long set that pass upwards to the clavæ of the medulla oblongata. Goll's columns are composed of fibres of this last set, and chiefly of those that spring from the posterior sensory roots of the nerves that supply the lower extremities, though perhaps also of those that spring from the posterior roots in the lower dorsal region. A degree of localisation is now possible within the limits of Goll's columns: for the innermost and hindermost portions of the columns are shown by Case 2 (in which the only symptoms were those of complete paralysis of the sciatic nerve) to be occupied by fibres that belong to the sciatic nerves. Pathology teaches us that Goll's columns have a much larger area in the cervical portion of the cord than Flechsig gives as a result of his embryological studies. Schultze remarks that the extent of the secondary degeneration is not always proportional to the extent and severity of the symptoms; which is probably due to the fact, that the area of degeneration is generally determined not by microscopical but by macroscopical examination, and this may easily lead to considerable error.

Descending degeneration was observed in the posterior columns in Case 5. Schultze has seen it in other cases, and Westphal, Kahler and Pick, and others have described it. It extends only 2 or 3 ctm. downwards. Schultze suggests that it is caused by degeneration of those fibres of the posterior roots that bend downwards as they enter the posterior columns.

Secondary degenerations in the lateral columns are both ascending and descending. Of ascending degenerations the most important is degeneration of the cerebellar tract. This was found after lesion of the lower dorsal cord, not after lesion of the lumbar enlargement and cauda equina. The tract as marked out by degenerations reaches further forwards that Flechsig represents it. Schultze has not observed the punctiform degeneration in the central parts of the lateral columns that Singer has described in dogs. In one case he noticed a partial degeneration of the anterior half of the column, but is doubtful whether this was a true secondary degeneration.

The author has nothing new to say of descending degenerations of the pyramidal tracts. In Case 5 there was descending degeneration not only of the pyramidal tracts, but to a less degree of the whole lateral columns, with the exception of their innermost portions (the lateral limiting layers), and also degeneration of parts of the ground bundles of the anterior columns: the disease extending 8 or 10 ctm. downwards.

The paper concludes with some remarks on the pathological anatomy of tabes, which Schultze regards as primarily an atrophy of the nerve fibres.

Westphal on Spastic Spinal Paralysis.—WESTPHAL (*Archiv f. Psych.* Bd. xv. p. 224) reports a case which presented the following symptoms:—Spastic paresis of the lower extremities, spontaneous twitchings of the muscles, heightened tendon-reflex, diminished sensibility to temperature, somewhat increased sensibility to pain; and the act of micturition was rather painful, and caused a powerful tonic convulsive movement throughout both lower limbs. Westphal remarks that the sudden forcible bending of a joint will often give us indication of an incipient spastic contracture, when slow, gradual passive movements tell us nothing. The case is remarkable, in that the symptoms remained stationary for about four years, when paralysis rapidly developed in the right arm, brain symptoms showed themselves, and death occurred in four weeks. It cannot be considered a typical case of spastic paralysis, on account of the bladder troubles and sensory symptoms.

The post-mortem disclosed primary degeneration of the pyramidal and direct cerebellar tracts of the lateral columns of both sides; slight irregular degeneration of Goll's columns in the cervical and upper dorsal regions; and softening of the central white matter of the hemispheres, particularly in the left cerebral hemisphere. There was hypertrophy and dilatation of the bladder. This case is in accord with Westphal's previous experience, that the symptoms of spastic paralysis may be associated with disease of the lateral columns, conjoined with disease of the posterior columns, if the latter does not extend to the lumbar portion of the cord.

Westphal returns to the subject of uncomplicated primary sclerosis of the pyramidal tracts of the lateral columns, and controverts Flechsig's statement, that the so-called cases of this affection in general paralysis, are really cases of descending degeneration of cerebral origin. According to Westphal, it is in cases of general paralysis, and in these alone, that this peculiar form of sclerosis has yet been found.

Vierordt on Combined Disease of the Anterior Cornua and Lateral Columns of the Cord.—Vierordt (*Archiv f. Psych.* Bd. xiv. p. 391) reports a case of which the following were the chief symptoms:—There was a gradual appearance of weakness in the muscles of the extremities, with subsequent atrophy of the muscles; there was the reaction of degeneration; there was no trace of muscular contracture; the patellar reflex was retained; sensibility and the functions of bladder and rectum were normal, and there were no bulbar symptoms. The patient died of an intercurrent attack of bronchitis. There was advanced degeneration of the grey matter of the anterior cornua of the cord; degeneration evidently more recent of the pyramidal tracts of the lateral columns from the lumbar region to the decussation, but not beyond this; degeneration in a moderate degree of the anterior mixed zones of the lateral columns; and degeneration of the motor nerves and of the muscles. The direct cerebellar tract and the lateral limiting layer were normal. Disease of the anterior mixed zone of the lateral columns has been noticed by many authors in cases of combined disease of the anterior cornua and lateral columns. It is also found in some cases of poliomyelitis anterior, and progressive muscular atrophy.

This case is an example of primary degeneration of the ganglion cells and nerve-processes of the anterior cornua, with secondary systematic degeneration of the pyramidal tracts. A remarkable feature is the absence of degeneration of the pyramidal tracts above the decussation. But notwithstanding the absence of bulbar symptoms, the case is closely allied to Leyden's amyotrophic bulbar paralysis. They are both characterised by degeneration of pyramidal tracts with flaccid atrophic paralysis, and so stand in contrast with Charcot's amyotrophic lateral sclerosis, in which there is degeneration of the pyramidal tracts, but rigid paralysis with contractures.

Hoffmann on Acute Ascending Paralysis.—Typical cases of acute ascending paralysis are characterised, according to Westphal and others, by an absence of morbid appearances in the cerebro-spinal nervous system; and the theory has been advanced that the disease is due to some toxic agency. On the other hand, cases have been described in which there was a well-marked affection of the cord and medulla oblongata; and this has suggested the view that the disease belongs to the group of acute myelitis. It is as a contribution to this discussion that Hoffmann reports his case *Archiv f. Psych.* Bd. xv. p. 149. Clinically, it was a typical cas[illegible] of the disease. Microscopic examination disclosed inflammatory changes in the cord and medulla oblongata. There was inflammation of the pia in the cervical and dorsal regions, with patches of myelitis, especially in the lateral and anterior columns, and small hæmorrhagic extravasations; similar changes were observed in the medulla, in particular in the pyramids and corpora restiformia. This case then supports the myelitic view of ascending paralysis.

Strümpell, Vierordt, and Müller, on Multiple Degenerative Neuritis.—The case that Strümpell reports (*Archiv f. Psych.* Bd. xiv. p. 339) was in its early stage diagnosed as a chronic poliomyelitis. There was extensive and rapidly developed paralysis in all four extremities, with marked atrophy of the affected muscles, and the reaction of degeneration; the muscles of the trunk were paralysed, the respiratory muscles and the diaphragm last of all; the functions of bladder and rectum were normal; there was no decubitus. The only symptom that threw doubt on the diagnosis was the slight sensory affection; there were in the early history of the case darting pains in the arms and legs, and during the latter stages analgesia, and at other times hyperæsthesia in the lower extremities. The presence of sensory symptoms will probably be an important point in the differential diagnosis of poliomyelitis and multiple neuritis. Other interesting symptoms were the persistently high pulse-rate, which was never under 120 per minute, and which may perhaps be attributed to an affection of the vagus, the œdematous swelling of the four extremities, and the commencing atrophy of the optic nerve.

The autopsy showed degenerative atrophy of the peripheral nerves and muscles. The anterior nerve-roots of the spinal cord were quite normal. The term degenerative *neuritis* is used, but there was no trace of even old inflammatory changes, and the disease is probably simply a nerve degeneration. The symptoms indicated that the motor nerves were implicated in a much larger degree than the sensory nerves; but the *post-mortem* does not throw any light on this point, which is deserving of special elucidation in future cases.

As regards the etiology of the disease, there is reason to suspect that it owes its origin to some infective source. Beri-beri, which is similar in many respects to the disease under consideration, has been shown to be a multiple neuritis, and it is endemic in many tropical and subtropical places. Tuberculosis is a frequent complication in cases of multiple neuritis. Strümpell thinks that many cases of acute ascending paralysis (Landry's disease) are really cases of multiple degenerative neuritis, and he points out the necessity of examining the peripheral nerves and muscles in all cases.

VIERORDT (*Archiv f. Psych.* Bd. xiv. p. 678) reports a case of this disease in a patient suffering from phthisis, and comments on the frequent association of the two diseases. He also carefully describes the different sensory symptoms, and indicates their relative diagnostic importance.

MÜLLER (*Archiv f. Psych.* Bd. xiv. p. 669) publishes another case, complicated with tuberculosis and mental symptoms.

Greiff on the Localisation of Hemichorea.—Two cases are reported (*Archiv f. Psych.* Bd. xiv. p. 598). The first was a case of left hemichorea following left hemiplegia, and the autopsy revealed lesions in the left cerebellar hemisphere, the right occipital lobe (implicating the occipito-temporal, lingual, and inferior temporal convolutions) and the right optic thalamus. In the optic thalamus there were two small hæmorrhagic foci, one in its upper and inner portion, the other at its lower border where it is contiguous with the pes pedunculi, into which the focus could be traced. Greiff concludes that it was this last lesion that caused the hemichorea by the excitation it produced on the pyramidal fibres of the peduncle.

The second was the case of a man, aged 51, who presented in the early part of his illness the symptoms of senile dementia, but afterwards epileptiform attacks developed, so that the case may be referred to the group of progressive paralysis (general paralysis). After one of the convulsive attacks, in which the left side had as usual been most affected, left hemichorea appeared, and lasted four days, when it declined into a hemiathetosis, and passed away. Microscopic examination showed, in an extreme degree, changes in and around the vessels with pathological alterations of the nerve-cells. These changes were most marked in the right central convolutions and the paracentral lobule. There was also a small focus of incipient softening in the upper part of the right pons adjacent to the pyramidal strands, with descending degeneration of these strands. It is impossible to decide which of these lesions determined the hemichorea, but in either case it was probably by irritation of the pyramidal fibres.

Unverricht on Epilepsy.—UNVERRICHT (*Archiv f. Psych.* Bd. xi p. 175) summarises the results of an experimental and clinical investigation into epilepsy under twenty-five heads. We can only notice a few of his conclusions. He distinguishes two varieties of the status epilepticus, experimentally produced: the oscillating variety, in which the spasm travels from one side of the body to the other, and then returns by the way it came, and so swings pendulum-like from side to side until the convulsion becomes general; and the variety in which there is a series of spasms commencing in one place and taking the same course, and following each other with ever-increasing rapidity, until they become general. The body temperature may be lowered to 34·8° C. without affecting the irritability of the cerebral cortex or the appearance of the status epilepticus. Anæmia and asphyxia only arrest convulsions when they are pushed to a degree that endangers life. Injections of chloral into the veins, or the inhalation of ether, readily arrests them. Extirpation of the various motor centres frees the corresponding muscles from participation in the convulsive seizures, though associated movements may still occur. Extirpation of the motor areas during the status epilepticus arrests the convulsions in the muscles supplied by these areas. Division of the corpus callosum does not prevent the spreading of the convulsion from one side to the other. Hæmorrhage into the ventricles arrests the convulsions on the opposite side of the body.

Zacher on General Paralysis.—ZACHER (*Archiv f. Psych.* Bd. xiv. p. 463) continues his paper on some peculiar forms of general paralysis (for abstract of first part of which, see 'BRAIN,' Vol. V. p. 566).

(1) *On the Tendon and Skin Reflexes during and after Paralytic Attacks.*—The author made observations on general paralytics with healthy spinal cords, with fascicular sclerosis of the lateral columns, and with combined disease of the posterior and lateral columns, respectively; and he comes to the conclusion that the tendon-reflexes during and after paralytic attacks are increased on the side which presents convulsive symptoms, even though a degree of paresis coexists, but are diminished or even abolished on the side which presents simply flaccid paralysis. If the tendon-reflexes were increased before the attack, they will either be still further increased or diminished, according to the above rule. If they were absent before the attack, they will not be re-established. A similar relation exists between the superficial reflexes and the condition of sensibility. Zacher accepts Schwarz's theory as to the correspondence between brain and spinal centres (see 'BRAIN,' Vol. VI. p. 417).

(2) *On certain Motor Phenomena during and after Paralytic Attacks.*—Muscular tension, rigidity, and contracture are often observed, and they probably express merely a different degree of the motor irritability that gives rise to convulsive phenomena. They are not to be looked on as evidence of spinal lesion, since they may be present where the cord is sound; they are probably produced by excitation of the cerebral cortex. Another motor phenomenon observed during these paralytic attacks is that of automatic, purposed co-ordinated movements, that have all the appearance of being directed by the will. Similar movements are familiar to us in some epileptic conditions and have been described by Fürstner in pachymeningitis. The presence of paræsthesia and ill-defined sense-impressions may account for the movements, or on the other hand they may simply be the result of a primary excitation of a motor area. Another of the motor phenomena alluded to by

Zacher is the conjugate deviation of the head and eyes, which follows the laws laid down by Prévost and Landouzy. And lastly, he draws attention to the occurrence of disturbances of the muscular sense, evidenced by the patient's want of knowledge as to the position of his limbs.

(3) *On Visual Disturbances during and after Paralytic Attacks.*—These may be divided into two classes. First, cases of pure mental blindness (*Seelenblindheit*). In the few cases Zacher has observed, the blindness was bilateral, though the motor symptoms were only on the right side. Secondly, cases of bilateral visual affection, which are probably examples of true hemianopsia, and are due to lesion of the posterior cerebral lobes. To these should be added perhaps a third class, including cases presenting a combination of these various visual disturbances, such as Fürstner and others have described. The existence of unilateral amaurosis of cerebral origin must still be regarded as unproved.

(4) *On some peculiar Vaso-Motor Symptoms.*—If a blunt object, such as a key, was drawn across the skin, a pale streak was seen, which soon became intensely red, and close to it appeared little square raised patches, which soon coalesced and formed a strip, slightly raised above the general surface. This is probably an exaggerated example of the phenomenon to which Trousseau drew attention, and which is exquisitely seen in some epileptic conditions.

Siemens on Refusal of Food in the Insane.—The author (*Archiv f. Psych.* Bd. xiv. p. 569, Bd. xv. p. 15) is of opinion that the refusal of food is by no means the serious symptom it is generally considered to be, and he makes the somewhat startling statement that abstention from food, if purely mental in origin, is attended with no danger, provided it is not prolonged over fourteen days, when no water is taken, or over fifty days when water is taken, and provided, too, that the loss of weight does not exceed 40 per cent. of the total weight. He is opposed to forcible feeding, except in cases of prolonged loss of consciousness and paralysis, on account of its mechanical dangers (injury to the fauces, pneumonia from food in air-passages), and also because it is unphysiological, taking no note of the digestive and assimilative capacities of the individual. He trusts chiefly to time, and tact, and the presentation of the kind of food the patient likes best. There is a class of cases, generally old hypochondriacal melancholic persons, which no sort of treatment, with the stomach-pump or without it, is able to save.

Langreuter on Paraldehyde and Acetal in Mental Diseases.—The author (*Archiv f. Psych.* Bd. xv. p. 1) recommends paraldehyde as a hypnotic in mental diseases. It is of most service in cases of epileptic excitement with feelings of terror, &c., and in the similar conditions of disturbed consciousness that occur in general paralysis. The superiority of the drug over chloral lies in its more rapid action and greater safety. The dose is from five to six grammes, and a sleep is produced in from five to ten minutes, that lasts about two hours, and often, especially at night, seven or eight hours. Acetal is similar in its action to paraldehyde, but it is not so reliable, and its smell and taste are much more disagreeable.

W. J. DODDS, M.D., D.Sc.

Wilks on Hemianæsthesia.—In vol. xxxvi. of *Guy's Hospital Reports*, Dr. Wilks makes a valuable contribution to the literature of this subject. He shows that, as is generally admitted, such cases do not admit of explanation by the hypothesis of a gross lesion; and gives reasons, moreover, why such lesion as there is cannot be in the region of the internal capsule. He emphasises the constant association of the phenomena of hemianæsthesia, hemianalgesia, &c., with the moral characteristics of hysteria; and counts this as one among other reasons for attributing what lesion there is to the cortex. He considers that the central circumstance in the malady is the arrest of the function of a large area, or the whole of the cortex of one hemisphere. Such cessation of action is common enough as affecting the two hemispheres simultaneously; as is evidenced by the occurrence of sleep, of stupor after blows, and other conditions. If we suppose such cessation to occur over part of the cortex instead of over the whole, it will account in great measure for the temporary one-sided losses and for the other manifestations attending them.

Gairdner on Lethargic Stupor or Trance.—An unusually well-characterised instance of this affection is related by Professor Gairdner (*Lancet*, Dec. 22, 1883, and Jan. 5 and 12, 1884). The patient remained in a completely helpless and apparently unconscious state from Aug. 1 to Nov. 18, 1883. During the whole of this time she did not once execute any movement save a slight movement of the eyelids. No response whatever was made to stimuli of the utmost justifiable intensity; and but for attentive artificial feeding the patient must have perished of starvation.

Such cases are probably closely allied to cases of hemianæsthesia. They may be looked upon as virtually cases of *double* hemianæsthesia, hemianalgesia and hemiplegia; and would, on Dr. Wilks's hypothesis, be looked on as due to loss of function of the entire cerebral cortex of both hemispheres.

CH. MERCIER, M.B.

Parant on Paralysis Agitans as a cause of Insanity. (*Annales Médico-Psychologiques*, July, 1883).—Dr. Parant relates the details of an interesting case of paralysis agitans, in which various manifestations of insanity appeared, and gives a résumé of what has been said by the chief French writers on this subject, by whom but little notice is taken of the mental side of the disorder in question; those who mention it at all, merely drawing attention to the dementia or general weakening of the mental faculties commonly supervening in the later stages. Professor Ball, however, has frequently seen mental disturbances in these patients, not only irritability and general mental weakness, but also true insanity with hallucinations and delusions, the insanity, in his opinion, generally assuming the form of melancholia, with delusions of persecution. Professor Ball and Dr. Parant both notice that when the tremor becomes less marked, the mental troubles also tend to disappear.

Dr. Parant compares paralysis agitans with miliary sclerosis and locomotor ataxy. He says, quoting Charcot, that in the cerebro-spinal form of miliary sclerosis, we generally, towards the end of the disease, find that the mental faculties become weakened, but that in some cases the classical forms of insanity present themselves. The sufferers from locomotor ataxy, also, occasionally become insane, the characteristic form here being melancholia with delusions of persecution and various hallucinations depending upon lesions of the optic, auditory, or other sensory nerves. The insanity in locomotor ataxy and miliary sclerosis is due to the extension of the morbid process from the cord to the brain; and Dr. Parant is of opinion that this is also the case in paralysis agitans. The writer thus formulates his conclusions:—

(1) That true insanity is occasionally met with in patients suffering from paralysis agitans; that it may present itself in various forms, but that mental depression is the predominant feature.

(2) That this insanity is probably due to the extension of the lesions of the cord to the brain, or to the formation in the brain of

isolated patches of disease springing from the same cause as the chief disorder in the cord.

Camuset on the prevalence of forms of General Paralysis in which Depression is the predominant feature. (*Ibid.*, May 1883.)—After examining a great number of general paralytics the author has come to the conclusion, that depression is more frequently met with in this disease than exaltation. He says that general paralysis presents itself under three chief forms: (1) Simple paralytic dementia; (2) paralytic dementia with exaltation; (3) paralytic dementia with depression; and on taking a general view of the cases of this disorder under his observation, he is struck by the fact that the patients are sad, melancholy and depressed; excitement, self-satisfaction and delusions of a grandiose character, being comparatively rare. He gives the result of his examination of 373 general paralytics in a tabular form, showing that cases characterized by depression are twice as frequent as those in which exaltation predominates.

R. Atkinson, F.R.C.S.

Bernhardt on Head-tetanus. (*Zeitschr. für Klin. Med.* Band 7, Heft 4.)—Patient, man of 32, had undergone, Oct. 17th, 1883, an operation for removal of a dermoid tumour on the left supra-orbitalis ridge. The wound healed quickly. On Oct. 22nd he had difficulty in opening mouth, and at the same time left-sided facial paralysis occurred, followed by pain in back and difficulty in swallowing. Tightness on the chest. Facial expression very peculiar, conditioned partly by the decided left-sided facial paralysis. Forehead smooth on left, and left eye open, nasolabial fold abolished, left corner of mouth drawn down. The movements of expression (*mimischen*), carried out on right side of face, did no take place on the left. In spite of left paralysis, expression of face strained and forced. Grinding of teeth loudly heard when he tried to open the mouth, due to convulsive contraction of masseters, which were tense and hard, especially on the left. In trying to swallow, convulsive reflex spasm was produced. Jaws firmly clenched, and muscles of swallowing spasmodically convulsive. The scar from the recent operation was one centim. distance from the left external corner of the eye in a slanting direction upwards, and was not particularly tender. Mind clear, behaviour anxious, pale face, speech normal, movements of eyes free in all directions, no squint, no double sight, pupils equal and moderate size.

Electrical excitability well marked with both currents in direct and indirect excitation of root and branches of the facial nerve on the paralyzed left half of face; with the same currents as those applied to healthy side, instantaneous contractions followed, except left frontal muscle which with direct excitation of the constant current, reacted to a weaker current than on the right side, but with distinctly slower contractions,

viz. Ka Sz = 1½° of the galvanometer,
Ka Oz = 3°, and A Sz by a current, not appreciated by the needle, but in striking contrast to the right side, where Ka Sz did not occur at 2½°, A Sz only by much stronger currents, and Ka Oz not at all.

In region of chin and lower lip there is slight loss of sensibility to touch. No rigidity or paralysis of extremities. Tetanus, or rather head-tetanus, diagnosed shortly before death, when involuntary convulsions occurred, and pulse soft, compressible, temperature raised. State of patient continued the same till death in hospital on 2nd November following.

Post-mortem.—Pia of brain had a cloudy appearance on convexity of brain, otherwise macroscopically nothing to be seen abnormal. Pons and medulla not yet examined. Pharynx and œsophagus intact. Some branches of N. fac. sinister, also of right facial nerves examined microscopically and found intact.

The region under scar on left upper border of left eye cavity, extravasated with blood.

The course of the disease here described agrees exactly with the description given thirteen years ago by E. Rose in his treatise on tetanus of a peculiar type, which he has called "Head tetanus, or tetanus hydrophobicus." These cases of tetanus according to Rose occur after wounds in the region of the 12 cranial nerves; a marked characteristic of this is the intense spasm of the pharynx, producing a strong resemblance to hydrophobia. Rose says "it is probable that these cases of head tetanus, tetanus hydrophobicus, as they occur particularly after damage in the region of the central nerves, allying themselves particularly with spasms of central nerves, and of the pharyngeal muscles, have misled eminent practitioners to identify tetanus with hydrophobia. No one, who has seen such cases of head-tetanus, would find a resemblance between severe hydrophobia and acute tetanus."

Besides these spasms of the pharyngeal muscles, the presence of the facial paralysis appearing on the same side as the original wound, especially stamped the disease as tetanus. Perhaps the affected nerve is swollen in the direction from the wound to the nerve centres: the paralysis would then be explained by the pressure in the unyielding fallopian canal. Rose has himself observed two cases of this kind, one ending in death, on which no light was thrown by autopsy, the other recovering.

In the above case the relation of the frontalis muscle to direct galvanic excitation was peculiar. The phenomena of degeneration-reaction was here shown: but it remains doubtful whether this reaction pointing to a change of the one muscle was dependent on a deeper affection of the facial root, or upon a local lesion of this particular frontal branch only occasioned perhaps by the operation. Dr. Gluck, the operator, stated that no section of nerve had taken place in removing the tumour; an exact electrical examination of patients in the second and third week of the disease would alone determine whether the facial paralysis in head tetanus is always slight (in the electro-therapeutical sense) or whether so-called intermediary forms occur.

Professor Bernhardt next refers to the "tension" of the features in tetanic cases, which has been remarked on by Romberg, Hesse and others. According to Koenig the patient's face expresses grief, or fatigue accompanied by a grinning appearance, according to the preponderance of the muscles of the upper or lower half of the face. The muscles of the middle portion of the face are more or less stretched by the powerfully contracting upper or lower muscles. Although Koenig does not refer to paralysis of the muscles of the face, he distinctly urges having observed these things in several chronic cases, the starting-point of which was a wound in the head or face. Rose further cites other cases of tetanus after head wounds, where besides spasmodic contraction of the pharynx, contractions of the muscles of the face with convulsive movements of the tongue occurred (case of Guastalla), and earlier still in 1853, Pflüger refers in his work, "On the functions of the spinal cord in vertebrata, &c.," to a boy with trismus and spasms of pharynx and face after a contused and incised wound of the inner part of right eyebrow, the trismus extending to the muscles of whole body, especially the right side, recovery ensued; also to a case of Larrey's, where tetanus came on 9 days after a wound to the right frontal region in a soldier, beginning in the right eyelid; and was relieved by cutting through the right orbicularis oculi muscle; another patient of Larrey's had tetanus hydrophobicus, after a wound of the nose, with persistent spasm of the face muscles.

Bernhardt adduces cases showing that contracture and paralysis can occur at the same time in the muscles of the affected side of the face, due to reflex spastic conditions although voluntary impulses are in abeyance. He considers that the motor branches of the trigeminus are strongly affected in every case of head tetanus, and follow Pflüger's laws, viz.: that in reflexes occurring on both sides of the body, the muscles most affected are found on the same side as the excited centripetal fibres. Sometimes tactile sensibility on the wounded side of the face is affected, analogous to the motor paralysis of the face; the nerves and muscles presiding over swallowing are particularly affected, hence the term of tetanus hydrophobicus of Rose.

Of 8 cases, 3 recovered completely after long duration.

Of 4 of the fatal cases, characteristic pathological changes in the central nervous organs were negative.

Bernhardt thinks that possibly changes in the facial muscles may be found.

Finally, he remarks that the term tetanus hydrophobicus or head tetanus does not sufficiently represent the disease which, in addition to the severe spasm of the pharynx, has, as its characteristic, the paralysis of the facial muscles on the same side as the wound, but he cannot suggest a better name.

He refers to two cases, one by Nankivell in the *Lancet* for July 14th, 1883, after a wound of the bridge of the nose, ending in death; the other by Bond in the *Brit. Med. Journal* for Nov. 10th, 1883, which recovered.

Bernhardt concludes that (1) all the cases do not necessarily end in death; (2) the cases of complete facial paralysis, which recovered, did not belong to the most severe forms; (3) the term tetanus hydrophobicus cannot be applied to all the cases, as spasm of the pharynx is not always present; (4) complete facial paralysis on the same side as the wound is an extremely significant characteristic of all these cases.

Franz Riegel on Spasm of the Muscles of Respiration (*Zeitschr. für Klin. Med.*).—A peculiar case of spasm of the respiration lasting a year. The patient had undergone varied treatment, until brought into the Klinik (Giessen), when a complete cure was effected in a few days.

Of the three types of spasm, viz. (1) affecting the inspiratory, (2) the expiratory muscles, and (3) complicated respiratory spasms, in this case the expiratory muscles were affected.

K. Q., 14 years old, of nervous temperament, fell into the water in winter time. Afterwards he complained of pressure and pain at the epigastrium—in 14 days' time difficulty in respiration came on. He complained of want of breath, difficulty in breathing, i.e. expiration. Internal organs and nervous system normal.

He had long pauses of breathing, of 20–30 seconds, followed by long inspiration and then long expiration, followed immediately by successive spasmodic expirations without intervening inspiration.

No diminution of thorax, but epigastrium violently drawn in and abdominal muscles severely contracted during the spasm.

Pressure on phrenic nerves and vagus produced no change in this breathing-rhythm.

Diagram of breathing taken by a canula inserted into the nostril.

First week after admittance to hospital, no treatment purposely given. No organic disturbance of any kind being found, and the boy perfectly healthy—a hysterical affection was suspected.

Psychical treatment resolved on. The boy was told the spasms could and must be suppressed—was made to breathe methodically and slowly—great improvement produced. To complete the cure he was told the application of the electric current was necessary. One application of faradic current sufficed to cure him. In 14 days' time no recurrence of spasms. This prompt success of a purely psychical treatment was a proof of the hysterical nature of the affection. Riegel further quotes Leyden's opinion, that spasm of respiratory organs is common in boys at the time when the voice changes.

Bernhardt on Partial loss of Sensibility (*Berlin Klin. Wochenschr.* 1884, No. 4).—The patient, a man aged 19, had gradual loss of feeling for four years, to pin-pricks, electric brush, heat and cold, in the right upper limb, the right shoulder, the right half of neck and head, bounded in front by the median line, behind by the middle of scapula and nape of the neck, above by the median line of head, and below by the fourth rib; the right side of the face (especially the forehead, nose, cheek, and lip) was very much less affected, this area being sharply defined by the edge of the lower jaw; the right ear, however, was as much affected as the right side of the neck. There was no motor paralysis, but some loss of power in the arms, and in the right more than the left.

No sign of hysteria. Legs often become numb when they are crossed. Organs of sense intact, on both sides. Power of both arms not quite so good as formerly.

There was a swelling on the posterior surface of the right elbow, he cannot flex the right elbow-joint properly, and crepitus is felt when the forearm is pronated and supinated; this is due to a fracture of the upper end of right ulna which occurred suddenly with moderate exertion, and subsequently the upper end of radius was dislocated from the ulna.

In the affected area tactile impressions are felt immediately, but not quite so clearly as in the left arm. Pressure sensibility and muscular sense normal; no ataxia. In the right upper arm pain for the strongest faradic current is abolished, but he feels the tension and drawing produced by the current. Pin-pricks felt as touches, but no retardation of impressions of touch. Mucous membrane of right conjunctiva, nose, and mouth, normal. Does not feel the pain of an electric spark on the affected area, and not appreciate boiling water there; but appreciates heat a little on the right face.

No atrophic changes except the fractures and dislocation which point to trophic changes in the bones.

Excluding other lesions, Bernhardt thinks that the lesion is in the right posterior roots of the cervical nerves from I. to VIII., or rather of all the posterior grey matter of the whole cervical cord, where the posterior roots enter.

Following Schiff's theory, that the tracts for transmission of pain are in the grey matter, and those for touch in the white matter of posterior part of cord, he thinks the disease involves also the great and small occipital, and the great auricular nerves, which come off from the cervical cord and supply parts of the ear and face, and also that the right ascending trigeminal root is affected.

He refers to similar cases of Kahler, Fürstner and Zacher, and Schüppel. If the disease should involve the medulla oblongata, as in the above cases and that of Westphal, phenomena of bulbar symptoms would occur, and disturbances of sensibility in the part supplied by the trigeminus. Bernhardt thinks, it is not always possible to diagnose during life, a gradual progressive lesion of the grey central substance of the cord from a syringo-myelitis; he believes that it is possible in some. He considers it very difficult to make a differential diagnosis, when the muscles are affected and not the skin, the lesion being confined to the motor parts of the cord. Syringomyelia has been found on post-mortem without any symptoms during life, and, similar to slow growing tumours of the brain, causing no symptoms.

C. E. Beevor, M.D.

BRAIN.

JANUARY, 1885.

Original Articles.

ON APHASIA.[1]

BY L. LICHTHEIM, M.D.,

Professor of Medicine in the University of Berne.

WHEN we read the recent controversies concerning the symptoms of Aphasia and their explanation, we might readily come to the conclusion, that there exist differences between the methods of investigation adopted by the chief authorities, and that it would be, therefore, desirable first to reach unanimity upon this fundamental question of method.

Happily such an opinion would be erroneous, and we can have no doubt as to the way to follow in our investigations, as there is no such divergence. The method which has hitherto yielded the results does not differ from those used in the natural sciences. Starting from the observation of facts, it culminates in the explanation of these facts. The correctness of our explanations must be subjected to the control of further observations. Precisely the same course is followed in experimental research, with the exception that, in our present subject, the experiments are not instituted at the will of the investigator, but are supplied to him by nature, and that he thus depends for them upon a happy chance. The erection of the building can therefore proceed at a slow pace only, and must rise by degrees as the result of many toilers' work.

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[1] This paper has been translated by me from the German manuscript; but our limits required that the original should be condensed. The whole task was by no means an easy one; the careful revision bestowed by Professor Lichtheim on the proofs, and for which I beg to return him my best thanks, has, however, materially relieved me from the responsibility I had assumed.—A. DE W.]

Nor do we meet with any divergence of opinion as to the end to be attained. Our task is to determine the connections and localisation of the paths of innervation subservient to language and its correlated functions. On the supposition of our having reached this end, we should then be able to determine the exact place of any solution of continuity in these paths, and account for its symptomatic manifestations with the same precision as we do for those of a motor or sensory paralysis depending on a lesion of the peripheral nerves.

Now, although all are agreed that we have by no means as yet reached this point, opinions differ as to how near we have arrived. We may, however, congratulate ourselves upon t simple fact of there being some agreement as to the fundamental meaning of disturbances of speech from cerebral causes. The amount of superstructure which will be raised on such a foundation must depend, in individual instances, upon the personal temperament of the architect. There is room both for the enthusiast and the sceptic, who both have their function to fulfil in the race for truth. The only necessary condition for the successful building up of the edifice, is that the one should not deny, the other not distort, acquired facts.

It follows from what I have already said, that every step which brings us nearer the fulfilment of the task before us must enable us to differentiate more accurately the clinical forms of aphasic disturbance. What to-day appears to us as a curiosity, as a case aberrant from the ordinary type, will to-morrow be classified as an instance of conformity to the law. This we shall find illustrated in the history of the previous researches into the nature of Aphasia. Broca was led, after many mistakes had been made, to bring into sharp relief aphasia in its narrower sense. Wernicke[1] was the first, to my knowledge, who distinguished between the latter symptom and those due to an interruption in the centripetal afferent paths. Besides these two chief forms, which he calls motor and sensorial aphasia respectively, Wernicke describes a third, and designates it as commissural aphasia (*Leitungsaphasie*). The following discussion, the object of which is to establish a further differentiation, will bear upon this triple division of Wernicke's, and give me the opportunity of mentioning the other categories of aphasia. I abstain from any further reference to the historical aspect of the question, which has been treated by Kussmaul[1] in a way which leaves nothing to be desired.

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[1] 'Der Aphasische Symptomencomplex;' Breslau, 1874.

The morbid types which I intend to discuss in the following remarks have been determined, in as far as they are new, deductively: it was the task of subsequent clinical observations to test the validity of the inferences. The necessity of differentiating still further the types of aphasia struck me on attempting to schematize the forms hitherto known, for the purposes of instruction. But I did not consider that my schema should be published until cases had been observed which coincided with the new types postulated therein. The important element of my task lies in the observations themselves and in their interpretation. Still I thought it advisable, in presenting my results, to follow the same path which I had myself trodden, giving in the first place the schematic representation from which I started in elaborating my views of aphasia. It will be seen that my conception is intimately connected with previous ones, especially with that of Wernicke: the points of difference will appear in the course of the argument.

The schema is founded upon the phenomena of the acquisition of language by imitation, as observed in the child, and upon the reflex arc which this process presupposes. The child becomes possessed, by this means, of auditory memories of words (auditory word-representations[2]) as well as of motor memories of co-ordinated movements (motor word-representations).[3] We may call "centre of auditory images" and "centre of motor images," respectively,[4] the parts of the brain where these memories are fixed. They are designated in the schema by the letters A and M. The reflex arc consists in an afferent branch *a* A, which transmits the acoustic impressions to A; and an efferent branch M *m*, which conducts the impulses from M to the organs of speech; and is completed by the commissure binding together A and M.

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[1] 'Die Störungen der Sprache;' Ziemssen's 'Cyclopedia,' vol. xiii. Leipzig, 1877.

[2] ['Wortklangsbilder;' or auditory word-impressions.]

[3] ['Wortbewegungsbilder;' or kinæsthetic word-impressions.]

[4] ['Klangbildercentrum;' and 'Bewegungsbildercentrum.']

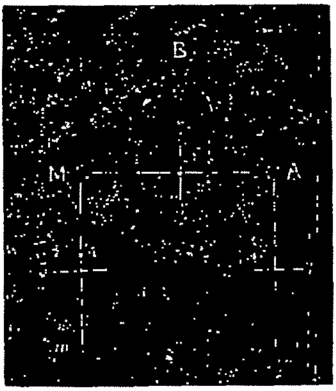

Fig. 1.

When intelligence of the imitated sounds is superimposed, a connection is established between the auditory centre A, and the part where concepts[1] are elaborated, B. All the diagrammatic representations of these phenomena agree so far, at least those given by German authors; beyond this, controverted points are found. The next step in the formation of volitional, or intelligent, speech involves a centrifugal connection between B and M. Our schema introduces, in consonance with most others, a direct commissure, whilst Kussmaul makes it pass from B through A to M. We shall soon return to this point. Leaving aside the point B for the present, we see from the diagram that seven different interruptions may occur in the paths therein assumed. It is manifest at first sight how variously the function of language may be disturbed by some of them; yet it is necessary, in order to gain a clear conception of the various types so produced, that we should include in our survey the disturbances of the functions of language involved in the acts of reading and writing. These are acquired in connection with the exercise of speech, and are hence intimately connected with it; the same nervous paths are, to some extent, brought into play. Reading postulates the existence of visual memories of letters and of groups of letters. We may learn to understand writing through the connection between such visual representations (centre O) and auditory representations: by spelling aloud we bring the auditory centre into action, and thus establish a connection, through the path O A, between O and B; in reading aloud, the tract O A M *m* is thrown into activity.

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[1] 'Begriffe.'

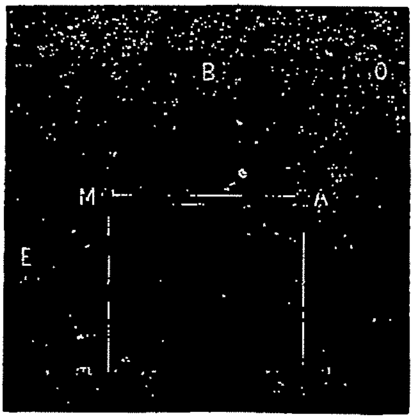

Fig. 2.

The problem with reference to writing is more complicated. The necessary movements have to be learnt, and associated with the visual representations; this is done through the commissure O E, designating by E the centre from which the organs of writing are innervated.

It is more difficult to determine the path through which volitional, or intelligent writing is executed. This tract must unite B with E, and clinical facts leave no doubt that it passes through M. There may be some doubt as to whether it leads directly hence to E, or passes round through A on its way thither. I shall return to this question presently; and adopt provisionally the former view, according to which Diagram 2 is constructed.

This figure makes it easy to derive the symptomatic type characteristic of each of the several possible interruptions in the reflex arc. I have been in the habit of using it for several years past in my lectures, and have found that it greatly facilitates to beginners the mastering of an otherwise very complicated subject. But if the schema is to have any value beyond that of an aid to teaching, it must be shown that the seven derivable types do really correspond to existing forms of aphasia, and that clinical observations are fairly reducible to them. I shall begin my discussion with an *exposé* of the several types.

1. Interruptions in M—the centre of motor representations of words, or motor centre of speech—give rise to the following association of symptoms:

Loss of (*a*) volitional speech;
(*b*) repetition of words;
(*c*) reading aloud;
(*d*) volitional writing;
(*e*) writing to dictation.

There still exist:—
(*f*) understanding of spoken words;
(*g*) understanding of written words;
(*h*) faculty of copying.

This constitutes the true "aphasia" of Broca, as well as the "motor aphasia" of Wernicke, and the "ataxic aphasia" of Kussmaul. The interpretation usually given of this trouble of speech is the same as that involved in my plan. It is this aphasia which rests upon the firmest basis, and I need not adduce examples from the large number of those on record, such as, for instance, the two celebrated cases of Broca's. I believe I have seen some pure cases of this description, but I have no notes of them, and their investigation was not conducted with the fulness of detail necessary to clear up the only point among the symptoms which still remains to be elucidated. I am referring to the question, whether there is understanding of written language in such cases. According to the diagram, the power of reading aloud should be lost, but that of silent reading preserved intact. I am sure that in uncomplicated instances this must be so, but I much regret that I have not had such a case to observe during the last few years; for the study of the literature of the subject has not afforded me any positive proof. In the majority of published cases no sufficient attention has been bestowed upon this question. It has indeed often been specified that the faculty of reading was intact: on the other hand, Trousseau[1] has shown that many 'aphasics' appear eager in their reading and yet do not understand what they read. He makes this observation precisely with reference to patients in whom the symptom of deficient understanding of writing was associated with the typical signs of Broca's aphasia. I shall return to this point, and show by personal observations how I think this contradiction may be explained.

2. If the continuity be broken at the point A, in the acoustic word-centre we find loss of:—

(*a*) understanding of spoken language;
(*b*) understanding of written language;
(*c*) faculty of repeating words;
(*d*) faculty of writing to dictation;
(*e*) faculty of reading aloud.

There is preservation of:

(*f*) faculty of writing;
(*g*) faculty of copying words;
(*h*) faculty of volitional speech.

This type corresponds with the "sensorial aphasia" of Wernicke, who has himself shown that though the faculty of volitional language is not lost, yet there are considerable disturbances in it. The latter is incorrect, inasmuch as wrong words are used, the words themselves are altered by the introduction of wrong syllables, occasionally to such an extent that the language becomes wholly unintelligible. This form has been called also "paraphasia." The explanation given by Wernicke of the fact, that in sensorial aphasia such disturbances occur in spite of the preservation of the tract for voluntary speech, appears at first sight rather forced. He assumes that the nervous influx descending along the path B M *m* sends a branch current to A, and that this *subconscious* innervation of the auditory memories of words secures the correct choice and expression of them; and that irregularities occur as soon as the co-operation of these elements ceases to take place.

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[1] 'Clinique Médicale,' vol. ii.

I accept this interpretation, but with a modification, namely, that the mere excitation of the auditory representation is not sufficient to secure correct speech, but that this representation must enter into relationship with the concept; that therefore the commissure A B must necessarily be intact for the same purpose. Paraphasia will be observed when in the arc B M A B, an interruption has occurred in such a way that language is not altogether arrested. The causes of such a modification will be pointed out presently.

It is by no means difficult to ascertain through self-observation, that such an innervation of the auditory word-centre does really take place. When speaking aloud, we cannot control the fact, because the words are actually heard, and the innervation of A from *a* being much more powerful, conceals that of A from M. But if we perform only the movement of the mouth necessary to the emission of a monosyllable, without issuing it, we shall be most distinctly conscious of the corresponding auditory representation. This observation has perhaps given rise to the assumption of Kussmaul, that the path from the concept- to the movement-centre passes through the auditory centre. This is one of the points on which his schematic representation differs from the one given above. His diagram corresponds to the one given here (Fig. 3), and his path for voluntary language would be B A M *m*.

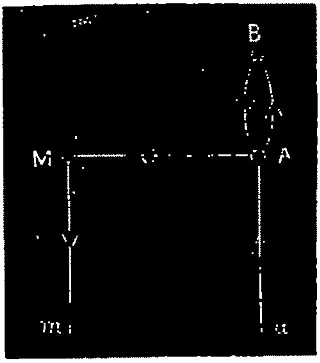

FIG. 3.

But it seems to me that this view does not correspond to the facts; the disagreement begins already in the case of Broca's aphasia, of which Kussmaul refers the seat, as we do, to the motor centre M. He assumes that the auditory representations are intact, and may be innervated from the concept-sphere; but this is, I believe, not the case. Trousseau, intending to prove that in aphasia words were forgotten, showed that in numerous cases the "inner speech" had disappeared. I have obtained evidence of this in a patient suffering from a form of the disease allied to Broca's type, and which will be adduced further on. It will generally be found a difficult task to elucidate this point satisfactorily; this is the method I use: I ask the patient to press my hand as often as there are syllables in the word to which an object corresponds. Those who have not lost the auditory representations can do this, even if their intelligence be limited, as I have been able to satisfy myself even under the least favourable circumstances. For instance, a patient who, besides a focal lesion of the right hemisphere, had had a hæmorrhage in the left half of the pons, and suffered, among other pseudo-bulbar symptoms, from complete speechlessness, preserved the faculty of fulfilling the test to the very last.

I have unfortunately, as was just said, not observed recently any pure cases of Broca's aphasia, but I have had a series of mixed cases in which this type predominated at least, and of pure cases of allied forms. I always found that the patients had lost the innervation of the auditory word-representations. These observations correspond so accurately to those of Trousseau, that I feel convinced that in ordinary cases of ataxic aphasia, the path from concept- to sound-centre must be interrupted.[1] Hence the diagram of Kussmaul cannot be correct, nor can a lesion in the path B A (Fig. 3) explain what occurs, for then the arc *a* A M *m*, and the power of repeating words would remain intact; whereas the latter, in Broca's aphasia, suffers as much as that of volitional language.

These considerations show that our schematic diagram may be considered as accurate, and that the co-operation of the auditory representations in speech is calculated to support the interpretation given by Wernicke of the paraphasic phenomena in his sensorial aphasia. I cannot refer to any typical instance of sensorial aphasia from my own experience; but Wernicke has given one,[1] carefully and minutely described, and in which the symptoms coincided[2] with the deductions from our schema.

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[1] A rare species of aphasia, included by Kussmaul under the ataxic, and to which we shall allude presently, differs in this particular.

3. The third form of aphasia, caused by the interruption of the commissure M A, has also been described by Wernicke, and corresponds with what we find in the diagram.

There are preserved:

(*a*) Understanding of spoken language;
(*b*) understanding of written language;
(*c*) copying words.

Paraphasia and paragraphia are present; showing, in accordance with what has been said previously, the existence of:

(*d*) volitional language; and
(*e*) volitional writing.

Similar disturbances are observed in the

(*f*) repetition of words;
(*g*) reading aloud;
(*h*) writing to dictation.

The path generally used in these acts (from A to M) is interrupted; still they are not completely in abeyance, because the tract A B M may be substituted for it. These actions, therefore, are not those of repeating, reading, and writing to dictation, properly so called, but are the result of impulses from the concept-centre; hence they manifest the same disturbances as volitional speech.

Cases of this sort do not appear to be very rare, though I am not acquainted with any observation of the kind as complete as could be wished. This is the more to be regretted, that it is precisely here that I have some doubts as to whether the construction of the path of volitional writing is correct. I have mentioned previously that I hold it as doubtful whether the path B M E be really used in volitional writing, or whether the channel of innervation be not from B through M and A to E (Fig. 4). The circumstance, that in volitional writing we are conscious of the excitation of auditory representations, does not prove that the *direct* innervation current really passes through the centre for the latter. The conditions are the same here as for the relation existing between these word-representations and volitional speech; whilst the chief current flows along B M directly to E, a secondary current may diverge from M through A to B, in order to secure the correctness of writing, as it does in the case of speaking. Clinical observations are necessary to decide on this point also; and the solution will be found in cases (described under 2) of Wernicke's sensorial aphasia, and of aphasia due to an interruption in the path M A. If localising the lesion of sensorial aphasia in A, we consider Diagram 4, the faculty of writing must have been lost too; whereas paragraphia only will be present, on the assumption that the relation of parts is as in Figure 2.

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[1] Loc. cit. p. 39.

[2] Excepting a difference in the power of volitional writing. This point will be considered in detail further on.

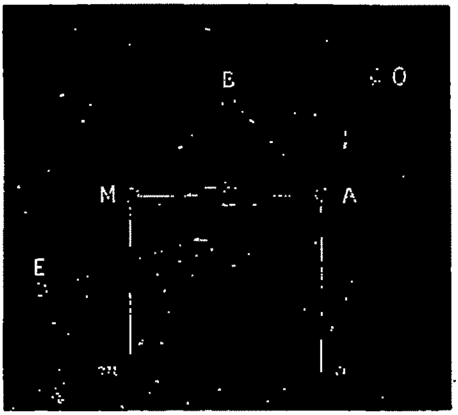

FIG. 4.

Though the question, when framed in these terms, appears a simple one, yet it is far from being easy to find an answer to it, because the published material at our disposal scarcely contains any sufficiently precise data to give us certainty on this point. The only case which we may adduce is the one already quoted of Wernicke's;[1] the patient could not write: "When sitting at the table to write, she takes the pencil presented to her point upwards, looks at it, turns it round, holds it properly, but makes only up and downwards strokes. When the pen is given to her in the same way, she turns it round, dips it properly into the inkstand, holds it properly, but with no better result." The agraphia persisted throughout, whilst the disturbance in the expression and intelligence of speech had disappeared. This case points to the correctness of Diagram 4; but it is not safe to draw a definite conclusion from a single instance, and unfortunately all other published cases of word-deafness fail us here, as will be seen on reading the thesis of Skwortzoff.[1]

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[1] Loc. cit. p. 39.

Precisely in the same way the form of aphasia arising from an interruption in the path M A, ought, according to Diagram 4, to be accompanied with a loss of volitional writing; whilst, according to Diagram 2, paragraphia ought to be present: but we cannot decide with absolute certainty from the observations at our disposal. In both of Wernicke's cases agraphia was present; whilst others, scarcely reported with sufficient accuracy however, leave room for doubts. In a case of my own the clinical aspects clearly corresponded to this type, whilst the post-mortem appearances coincided with Wernicke's description. I relate it here, because such autopsies are rare; but, owing to the insufficiency of the notes, it cannot serve in deciding on the point we are discussing.

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[1] 'De la cécité et de la surdité des mots dans l'Aphasie,' Paris, 1881. On page 84 a Case of Broadbent is mentioned which could be opposed to that of Wernicke. It is one apparently of sensorial aphasia, for besides imperfect understanding of language, the speech consisted in a kind of unarticulated jargon. It is said that the writing was not involved. But apart from the fact, that it is doubtful whether this was really volitional writing, and not only that to dictation or to a copy, the whole description is given in such a fragmentary manner, that we cannot tell whether we have to do with true sensorial aphasia, or some variety of it. The case, therefore, cannot be brought as evidence on the subject before us.—Since this paper was in the press, I read a typical case of Wernicke's sensorial aphasia described by Grasset (Contribution clinique à l'étude des Aphasies. 'Montpellier médical,' Jan. 1884) and in which also there was complete agraphia. This case too, therefore, points to the correctness of Fig. 4.

CASE I.—*Right hemiplegia; aphasic disturbances.—Obliteration of the Sylvian artery; brown softening of the left insula and neighbouring portions of frontal and temporal convolutions.—Pachymeningitis interna.—Valvular heart-disease, &c.*

J. S. B., aged 46, a labourer in Thun, was brought into hospital at Berne on the 3rd of April, 1883. We failed to obtain any clue to the beginning and course of the disease.

Sensorium normal. *Speech* much altered. When asked to relate his history, he strings together in a fluent manner numerous words, of which scarcely one now and then can be made out. The following were noted: "Evening, five and twenty, and." Patient is aware of the incorrectness of his diction, and tries to assist himself by gestures. He succeeds better with single short words and answers; thus, in answer to the question, "What was there for supper?" he answered, "Bread, meat, potatoes," with only two mistakes. His own name he mutilates.

Repeating.—When he repeats connected sentences he manifests the same defects as in volitional speech; single short words are pretty correctly rendered. No note taken of the way the patient names objects.

Understanding of Speech entire.—*Understanding of Writing*, whether printed or hand-written, is preserved. On reading aloud he makes the same mistakes as in talking.

Writing very imperfect. He mixes up the order of the letters in a word; and usually stops after an attempt of short duration. He can copy what is set before him with absolute correctness.

Motility.—Slight paralysis of the right arm and leg; apparent on simultaneous action of both sides of the body. Paresis in buccal region of right facial.

Sensation normal. The tongue deviates to the right, and is the seat of tremors. We pass over the symptoms in the chest and other organs. Symptoms remained unaltered until the beginning of May. Then consciousness became obscured during the next few days, and he died in the night of the 8th to 9th of May.

The autopsy was made, and a full report drawn up, by Professor Langhans. The brain presented the following appearances:

Inflammatory membranous deposits on the inner surface of the dura-mater on the left side. Pia-mater somewhat opaque and whitish, especially over ascending, and apex of temporal convolutions, where it is adherent.

A good deal of serum at the base of the brain. There is a considerable depression of the convolutions bordering the upper and posterior part of the left Sylvian fissure, as well as of the ascending frontal and parietal convolutions. The Island of Reil is sunken, forming a depression into which the second frontal convolution falls suddenly to a depth of 1½ ctm.; the third (lowest) frontal convolution terminates into it likewise. Here the pia is inflamed with yellow discoloration. The depression is bounded posteriorly by the fissure of Sylvius. The middle portion of the first temporal convolution is somewhat sunken opposite the depression of the Insula, in which the consistence of the cerebral matter is soft, with harder patches around.

It is difficult to follow the Sylvian artery beyond the point where it reaches the Insula, and presents on the length of 1½ ctm. a tough whitish appearance; it is occluded just before the division and a short way up the posterior branch.

The softened patch occupies the bottom of the Sylvian fissure and extends to about 1½ ctm. of the posterior part of the inferior frontal convolution, and to the neighbouring portions of the ascending frontal convolution. A fragment of the cortex of the ascending temporal is also wanting, but there is no yellow discoloration here.

The case just described is undoubtedly one of the third kind of aphasia; the symptoms tally with the theoretical postulates established previously. But the point which to us is most interesting, the capacity of the patient for volitional writing, is not established in the notes, because it is not sufficiently distinguished from writing to dictation.

In the absence of more definite data than those already adduced, whereby to decide which course the impulses follow in volitional writing, it appears to me more probable that Diagram 4 gives the more correct construction. There is one difficulty, however, and it is that in Broca's aphasia, writing to dictation is disturbed, a fact not deducible from Diagram 4, but with which Diagram 2 appears more consonant. On the other hand, as we shall see, in some cases where volitional speech is lost, but not the faculty of repeating words, the first syllables only are repeated correctly; when the patient is to repeat a sentence it is necessary to say it word by word. The explanation of this phenomenon is easy. If four or five words are given together, the patient must retain them in his memory, and thus what he says ceases to be a repetition, properly so called, but is an effect of volition. This is still more the case with reference to writing, an act which takes a much longer time; but few letters are written really to dictation, and indeed one finds in many cases of Broca's aphasia that the first letter only of a word can be written down, the rest of the word being unreadable.

Further observations cannot fail to throw light on the question. This controverted point does not apply to the remaining forms of aphasia.

4. A variety of motor aphasia is created by interruption of the path B M, of which we have many examples. From the diagram we should expect the loss of

(*a*) volitional speech;
(*b*) volitional writing;

whilst there are preserved—

(*c*) understanding of spoken language;
(*d*) understanding of written language;
(*e*) the faculty of copying.

So far the symptoms coincide with those of Broca's aphasia. They differ inasmuch as there is preservation of

(*f*) faculty of repeating words;
(*g*) writing to dictation;
(*h*) reading aloud.

As an example of this kind of aphasia, characterised by loss of volitional speech and preservation of power of repeating words, I may mention a case of Hammond.[1]

Still more striking is the faculty of reading aloud. Most recorded cases are incomplete in this respect. I therefore give a case of my own which beautifully illustrates this point.

CASE II.—*Traumatic aphasia.*

Dr. C. K., a busy medical practitioner, had a carriage accident, and was carried home unconscious. Three hours afterwards he was bled, when consciousness returned. There were severe bruises on the right side of the body, and of the head, which was ecchymosed. The movements of the right arm and leg were never absent, but weakened, whether through paralysis or injury is not certain. Immediately after venesection the right hand could be stretched firmly, only lifting it was difficult. Sensation normal. Difficulty of swallowing during the first two days; fluids readily got into the air-passages; mouth not easily opened; tongue difficult to pull out. Patient got up after about a week, when it was noticed that he dragged the right leg and swayed a little.

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[1] 'Diseases of the Nervous System,' 7th ed., chap. VII.

Speech was much affected: the first day he only said "yes" or "no," but quite appositely. Gradually, more and more words returned, at first imperfect. Whilst his vocabulary was still very meagre, it was observed that he could repeat everything perfectly. Soon after the accident he began to read with perfect understanding. It was established beyond doubt that he could *read aloud* perfectly at a time when he could scarcely speak at all. The statements of his wife are most positive and trustworthy on this point, though he himself does not remember what took place just after the accident. She states that after much difficulty in making himself understood by gestures, he obtained a newspaper, and to the great astonishment of all present he began to read fluently. She herself thought it most strange and inexplicable.

He could not write voluntarily at all; but this faculty returned slowly and imperfectly, as did speech. On the other hand, he could, soon after he had left his bed, *copy and write to dictation.* He spoke German and French fluently before the injury; but German rather the better of the two. As the aphasia diminished, German words returned before French.

Immediately after the accident the right pupil was dilated and immovable; no troubles of vision. I saw the patient six weeks afterwards, at a time when the loss of speech had to a great extent disappeared. One could observe a slight paresis in the right lower facial region; some weakness of the right leg, but nothing noticeable in the right arm. Sensation was normal, except that he did not so readily recognise objects with his right as with his left hand. The right pupil sluggish; no disturbance of vision nor hemiopia.

In every other respect he is in a normal condition, with the exception of speech. His vocabulary is copious, but he does not talk much, and speaks in a drawling manner. From time to time he misses a word or construction; he then tries to express himself with gestures. Speech much more defective when he must name objects shown to him; then many names escape him, and he also makes mistakes (*e. g.* for "Bild," he says "Milbe," then corrects himself and says "Portrait"; for "Stahlfeder" he says first "Bleifeder," then "Tintenfeder"). Many words are missed in

French also; he finds the French equivalents of the words he can say in German. Patient says himself that the auditory representations of the words he cannot find are missing; he cannot tell the number of syllables in them. If these words are spoken to him or written before him he says them at once, but forgets them immediately. He repeats correctly whole sentences, if not too long.

He understands spoken and written language perfectly. He can read aloud with the greatest fluency, and with scarcely any stoppage. Writing very imperfect. Asked to write the history of his illness, he puts down mutilated, meaningless words, in which it is impossible to discover any sense. Writing single words, names of objects shown, gives a somewhat better result. There is a parallelism here between writing and speech; such names as he cannot say, neither can he write.

On dictation, he writes fluently and without faults. The strokes are somewhat clumsy. He copies equally well. Intelligence normal. His wife assures me that he manages business perfectly, thinks of things in time, &c.

I saw the patient again a month later. Great improvement in his speech, which is fluent, and with almost no hesitation; he names objects with much less difficulty; words are rarely mutilated. Writing is still deficient, though much improved. What he writes is perfectly legible.

5. Still better known than the preceding is the type of aphasia arising in an interruption of the path M *m*. The diagram indicates loss of:—

(*a*) Volitional speech;
(*b*) repetition of words;
(*c*) reading aloud.

Preservation of:—

(*d*) Understanding of speech;
(*e*) understanding of writing;
(*f*) faculty of copying.

Again, and as a distinguishing feature between this and Broca's type, there remain:—

(*g*) Faculty of volitional writing;
(*h*) of writing to dictation.

There are numerous examples of patients who had lost the faculty of speaking, and could yet make themselves understood by writing. I have seen several cases of this description, and possess the notes of one in which the patient was under my care several years ago, and wrote his history himself. But it would be useless to relate it here, in presence of the material already at hand.

According to Kussmaul, such cases present the true uncomplicated type of ataxic aphasia, that is, of that form of speechlessness, "in which patients, with freedom of intellect and of movement of the tongue, have the memory of words as acoustic signs, but are unable to emit them. The proof that they really possess these signs is found in the fact, that they can embody them in writing. When asked to articulate them as sounds or words, they cannot, even if they are shown how to shape their tongue and lips."

It is obvious that this description includes the type now before us, and Kussmaul gives a series of most pregnant instances in his chapter on ataxic aphasia. But the aphasia he so designates extends over a wider range of cases, such as those of Broca's aphasia, which we have discussed under our first heading. In these cases there is ataxic agraphia also, owing to the implication of the centre of co-ordination for written signs, which is distinct from, though connected with, that for spoken signs. The close relationship between these centres explains the fact, that the two symptoms so often co-exist.

Kussmaul's views rest on the assumption that, as the motor tract of spoken language is innervated by the left hemisphere only, so is the tract of written language; for it is only under such circumstances that a lesion of the left hemisphere could cause agraphia by an injury to the writing centre. I cannot accept this interpretation, but hold it more probable that the innervation of the movements of writing originates in both cerebral hemispheres.

When we learn how to write, we apparently employ the left hemisphere only; but we must assume that the right hemisphere is the seat of slight nervous action as well, for it can be shown that a certain amount of facility is also imparted for the same movements with the left hand. When the left hand executes "mirror-writing," it performs the same movements as the right hand in ordinary writing, and it is easy to show that the left hand writes mirror-wise better than it does the usual type. In my own case, even, the strokes are more correct if I write in English characters. Now it could be objected that this innervation of the left hand flows from the left hemisphere; but this is improbable, for in some lesions of the left hemisphere it happens that this tendency of the left hand to execute mirror-writing becomes manifest. Moreover, the fact, that in complete right hemiplegia the power to write with the left hand is not diminished, is opposed to the unilateral innervation of the movements of writing. Therefore the point E, which in our diagram represents the centre of writing movements, must not be looked upon as single; in addition to the point E in the left hemisphere, there is a corresponding spot, E′ in the right, to which commissures go, and which can come into action when there is some lesion of E. There are other proofs that in agraphia the motor innervation of the movements of writing is not involved. In pure cases of Broca's aphasia the faculty of copying is preserved,[1] whilst that of reading aloud, and of every kind of repetition, is lost. On the other hand, these patients are not only unable to write, but cannot even compose words with letters placed before them. The real cause of the agraphia does not depend on any lesion of the motor apparatus, but on the impossibility of

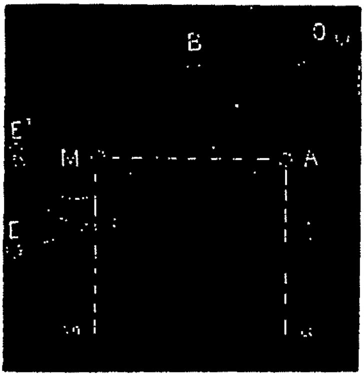

FIG. 5.

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[1] See example given by Wernicke, loc. cit., p. 56.

reproducing voluntarily the auditory representations.[1] I have already shown how one can convince oneself that such patients are wanting in the faculty of volitional recall of the auditory representations, and have stated how far I had reached certainty on this point.

Since the above has been written, a case of Pitres[2] has been published, which gives direct evidence of the correctness of my views on agraphia. The patient was suffering from agraphia, the residual sequel of an apoplectic seizure; speech, understanding of language and writing were intact, whilst volitional writing and writing to dictation were impossible, the faculty of copying being preserved. The lesion would be referred by me in the path A E (Fig. 4). The interesting point about the case is, however, that this agraphia affected solely the right hand; the patient could write correctly with the left hand, and copy with his right what he had written with his left hand. This shows the correctness of the diagram, in which the centre E is represented as existing in both hemispheres, and the path A E as being double.

It is obvious that this agraphia has nothing in common with that occurring in Broca's aphasia. As could be expected, the patient could make up words from their component letters, a feat impossible to those suffering from Broca's aphasia.

Pitres adduces two more cases of isolated agraphia. The first, quoted from Ogle, is apparently much more complex. The patient could not even copy, nor make up his name with the letters, and had serious embarrassment of speech. The lesion was probably a more extensive one, and took its origin in an injury received eight years previously. In the second case (from Charcot) the conditions were likewise less simple. We are not told whether the agraphia was bilateral. I may add, that the paths A E and A O may be interrupted at the same time, as they appear (see Fig. 5) to run together in the brain for some distance before they separate to go into the two hemispheres. Isolated agraphia will therefore, under certain circumstances, appear as bilateral.

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[1] We learn to write with the co-operation of auditory representations, hence, we cannot write without it. Every lesion of the path B M A (Fig.) 4 destroys the innervation of the auditory representation from the concept-centre, and must necessarily result in agraphia.

[2] "Considérations sur l'agraphie à propos d'une observation nouvelle d'agraphie."—'Revue de Médecine,' 1884, 11.

The theoretical types with which we have hitherto been engaged corresponded closely with well-known varieties of aphasia. There remain two, due to lesions of the commissures A B and A *a*, which must present themselves as cases of sensorial aphasia. Owing to the rarity of examples of the latter, it would have been but natural had I had no actual examples of it to adduce, though it would obviously have weakened my whole position, the full confirmation of which requires a filling up of every gap. I have, however, found the two missing types, and their entire correspondence with the theory of aphasia here developed is a warrant for the accuracy of the hypothesis.

6. Lesions of the path A B, would give rise to the following symptoms. Loss of:

(*a*) understanding of spoken language;
(*b*) understanding of written language.

There are preserved:

(*c*) volitional speech.

For the reasons stated above, there will be paraphasia. So far, the symptoms coincide with those of Wernicke's sensorial aphasia. Here, however, there subsist,

(*d*) volitional writing,

which will also have the characteristics of paragraphia;

(*e*) faculty of repeating words;
(*f*) of reading aloud;
(*g*) of writing to dictation.

Owing to the interruption of communication between A and B, there must be a complete loss of intelligence for what is repeated, read aloud, written to dictation by the patient.

There also subsists the

(*h*) power of copying words.

I shall now relate a case of my own which presents all these characteristics.

Case III.—*Aortic insufficiency. Cerebral softening. Sensorial aphasia.*

J. U. Schwarz was admitted into the Inselspital on the 19th of May, 1884. Sixty years of age—nothing noteworthy in antecedents.

He was well up to the 15th of May. The next night his wife noticed that he became restless, spoke in his sleep, rose and returned to bed, &c. At 4 in the morning it became apparent that something had happened; what he said had no sense. He did not answer to questions; he tried very hard to make himself understood, and the same mutilated expressions were constantly repeated. His wife thought he had lost his reason. His apparent intention was to complain of severe pains in the forehead and nape of neck. This state of things went on, and as he was losing strength, he was taken to the hospital.

Patient is a rather fat, pale, man. Features flabby; expression dull. On admission, he gave the impression of a subject with psychical deficiencies. He talked a good deal, but repeated the same phrases. He points to his head and says, "Oh how stupid I am, I cannot," &c. It is difficult to make anything out of him, for he understands little that is said to him; but it is easy to ascertain that he is in possession of his intelligence, and that the apparent psychical deficiency consists only in his difficulty in understanding others, and in making himself understood. His actions leave no doubt on this point.

Spoken language.—The disturbance in his power to understand it is very evident (verbal deafness). If one stands behind him and talks to him, he turns round and asks: "Do you speak to me?" The simplest requests, to show his tongue, shut his eyes, are answered by: "I don't know what one wants."

Speech.—There is no deficiency in his vocabulary. He talks a good deal in a flowing manner: he seldom is short for a word; he occasionally uses a wrong or a mutilated one. But this is unfrequent, and in this respect he has greatly improved on what his wife described he was. He is in great difficulty when he has to name objects shown to him; he finds the names with the greatest difficulty, and assists himself with descriptions. Instead of "wine," he says "that is strong;" for "water," "that is weak," &c.

Repetition.—He obviously could repeat correctly all that was spoken before him; but he apparently did not understand what he did repeat, as for instance the words, "My name is Peter Schwarz, and I am already 4 years old," which did not draw any signs of denegation on his part.

Written Language.—He understands nothing printed, or handwritten. The simplest things placed before him he cannot read nor decipher. He knows the name of most letters, and gives it correctly; he only confounds the capital I.

Reading aloud.—He can make up letters into words, and he can read aloud by spelling; but it is evident that the sense of the word remains closed to him. Out of the sentence, "Will you have a glass of wine?" he goes correctly as far as "you" but has manifestly no idea of the meaning.

Writing.—His volitional writing is worse than his speaking—but he does write a few words correctly. In "Meine Frau" (my wife), he manages the first word, not the second.

Writing to dictation was not tested at the very beginning. Later it was found that he could do it well if each word was given singly; whole sentences are rendered inaccurately, words being missed.

Copying.—He copies perfectly, changing German into English characters, but does not understand anything of what he so writes.

Motility and Sensibility.—No difficulty in walking or standing. Movements of arms and legs quite normal; sensation likewise.

Reflexes.—Skin reflexes normal. Patellar and Achillis-tendon jerk absent.

Cerebral nerves.—1. Smell normal.—2. Nothing noteworthy.—3, 4, 6. Movements of the eye normal. Pupils small, react to light.—5. Normal.—7. No weakness of facial muscles.—8. Hearing not very acute; but present on both sides; he hears the tick of the watch only when close to the ear.—9. Taste appears to be normal. He can recognise and name acid; seems to recognise sweet, but cannot name it. He calls bitter "strong." Deglutition normal. 10. Voice natural.—11. Movements of the tongue free.

In the further evolution of the case, the word-deafness receded without disappearing altogether; on the 21st of May, the improvement was already very marked. On the 22nd the following conversation was held. "Shut your eyes."—He does it. "Take this glass of wine from the table."—He turns round and says: "Are you saying anything to me? I don't understand it." "Do you like red or white wine best?" "Here is some red wine." "Put your right leg out of the bed." "What shall I take?"—and he puts out the left. "Put your right forefinger to your nose."—"Some thing right? What must I do?" He puts the right leg out of bed. "Drink the Professor's health with this wine."—"As you wish. . ." The request is repeated. "Shall I take red wine for me or for you?" "Show your hand."—He does it. "Do you cough much?"—"Only to-day, as the weather is so bad." "What costs the cheapest wine you buy?"—"I don't know the words any more," &c.

His writing improves also. On the 21st he could write the alphabet easily. German and English characters are used promiscuously. On the 29th he made the first attempt, on being asked, to write a letter.

"*Ich soll*[1] Herrn Professor etwas wissen, *aber was soll ich etwas gheides wissen.* Ich weiss garnichts davon."

He still fails to make out written language; but this does not hold for numbers. He reads the multiplication table, and tells the results, though with a little uncertainty.

At the beginning of June he understands what is said to him much better; his speech is to a slight degree paraphasic. But one notices now that conversation is easier, that he has difficulty in finding the names of objects shown to him, and that he immediately forgets them when they are told. Thus, he was shown a knife and asked "What is this?"—"I have seen something like it, but I can't remember the name." "Is it a knife?"—"Yes, knife, knife!" "What is this?" showing him a cap. He mutters, but cannot find the word. "Is it a coat?"—"No," pointing to his own coat. "Is it a cap?"—"Yes, a cap." "Tell us what you have been eating."—"Eaten something, this morning at 8 o'clock drank something, and again at 12 o'clock, but I can't tell more." "Take your pocket-handkerchief, and wipe your spectacles." Patient does it. "Take my purse, take five francs out of it, and give them to So-and-so." He takes the purse, and five francs out of it, but does not understand the rest.

His understanding of written language is very bad still, though when he reads aloud he makes out the meaning better, a fact which corresponds with his improvement as to spoken language. When he is not allowed to read aloud, he apparently spells inwardly.[2] Even what he copies to dictation is obscure to him. I dictated to him an I. O. U. for 20,000 francs, which he wrote down, and allowed me to put in my pocket without giving the least sign of emotion. Volitional writing is still accompanied with well-marked paragraphia, though there is improvement in this respect. He wrote the following letter to me on the 4th of June:—

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[1] [The words in *italics* were written in English characters, the rest in German. The word "wissen" is apparently used for "schreiben." "Gheides" is meaningless.]

[2] When questions were asked, he, at the beginning of his stay in the hospital, used to repeat them, it seemed to facilitate his comprehension; but occasionally did not understand them even after repeating them.

"Juni Mittwoch d. 4ten.—Herr Professor Dr. Lichtheim. Ich möchte heute etwas schreiben. Ich weiss noch sehr wenig. Ich bin Arbergergasse No. 52, mit meiner meiner Lieben um 1 und $\frac{1}{2}$ Stunde sehr gern geschen. Ach wie tausend Mal so gern für seine liebes Herz. Ach gott wie gern sollte ich viel mehr wissen und mein liebes Herz müsste mal allte Tag, oft die weiss täglich hundert mal und ich weiss fasst noch nichts mehr was ich zu wissen sollte. O wie viel zu wenig daheim. Ich will am morgen Mittags daheim Arbergergasse zu meine Liebenfrau. Fünf mal heute noch. Ich will auch noch etwas schreiben.—Schwarz-Beer."

Patient left the hospital on the 6th of June, but came to see us once every week. Every time he showed further improvement. On the 23rd the following notes were taken:

1. *Volitional speech.*—Paraphasia is very slight; word-amnesia, on the other hand, is considerable, though not so apparent in current conversation. He remembers the names of objects a little better; still he is very deficient in this respect.

2. *Volitional writing* is improved. There is less paragraphia.

3. *Understanding of spoken language* is much better. Patient understands almost everything he is told, even the longer sentences. Complicated tasks are executed correctly. Still, beyond a certain point, he still manifests deficiency in this respect; and it is impossible to appeal to his self-observation.

4. He *repeats words* correctly, and in longer sentences. He does it more quickly and intelligibly.

5. *Reading aloud.*—More ready; but he still mainly does it by spelling. He understands better.

6. *Understanding of writing* more deficient than that of speech, though unquestionably improved. He understands short words at once. Longer ones take him an interval, during which he seems to put them together; he used to repeat them, but this being forbidden he appears to spell them inwardly.

7. *Copying and writing to dictation* are carried out properly. He understands much of what he writes. The experiment with the I. O. U. does not succeed any longer.

The last time the tests were applied was on the 25th of July. He still talks somewhat slowly, the arrangement of words is often peculiar, but it takes time to discover that words are wanting in his vocabulary. He is easily stopped by proper names, and finds them with difficulty. The word-amnesia, however, is much less marked.

He seems to understand all that is said; one is occasionally obliged to repeat a question; and he himself repeats, as if with astonishment, proper names. Much paragraphia still present when he writes of his own accord, though a letter he wrote to me on that day shows a marked improvement. Progress is also noticed in the other respects.

On the 30th of July I was called to see him. About midnight his wife noticed that he suddenly became restless. He tried to get up, and fell. The left arm appeared to be paralysed; but he can give no account of the state of the sensorium. He has been restless since, and talked nonsense.

I found his consciousness slightly affected; he is continually babbling as a delirious fever-patient. He reacts a little when called, but does not recognise people about him.

The eyes are chiefly turned to the right; it is difficult to make him fix them; when called he answers "yes," but turns them more to the right, and does not follow the finger. Pupils rather contracted.

He appears to have headache on the right side, points to it with his hand, and applies a sponge to it. Left arm flaccid and paralysed; left leg apparently sound, yet on rising he bends towards the left side.

Left naso-labial fold somewhat effaced. Sensation and reflexes on the left apparently diminished. No paraphasia noticeable in what he talks. There was constant vomiting; and the patient was taken to another hospital, owing to my removal into other wards, and is still living.

A few remarks on this case, in which the symptoms agree with those postulated by an interruption in the commissure A B, may not be out of place. It may be asked why he spelt the words, though the path O A M *m* was intact. I think this is explained by the fact, that for fluent reading it is necessary to understand the words; we guess the words before we have actually read them through, and gain much time thereby. When we have to say long words in a foreign language, we also must spell to a certain extent. Our patient shows also a phenomenon which has been observed in other cases of the kind; he repeated the question of which he did not understand the meaning. This condition is closely allied to the *Echolalia* of Romberg; and Spamer,[1] who also deduced the possibility of this symptom from his schema, used this name. In our case it did not appear as if the repetition necessitated the understanding of the words heard, by means of a centripetal path[1] from M to B. The circumstance that the patient supplied his lost understanding of written language by reading aloud, must be explained differently. The case shows, with many others, that disturbances in the use of written language are much slower to disappear than those connected with speech. Paragraphia here persisted much longer than paraphasia, and the patient understood writing but very imperfectly, whilst verbal deafness had practically passed away.

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[1] 'Ueber Aphasie und Asymbolie;' 'Archiv für Psychiatrie,' vi. 523.

This may be explained by assuming, that the recovery of the understanding of words is not effected by a new connection between A and B, but that the sound-representations are deposited at another point A′ which is connected with M, B, and *a*. We should thus obtain a state of things as represented in Diagram 6, where the destroyed path A B is not shown, but where the vicarious connections are depicted by dotted lines. With the assistance of the figure, one easily sees that the patient can easily understand by the ear what he reads aloud; whilst silent reading remains senseless. Silent spelling is already of assistance under these circumstances, because along

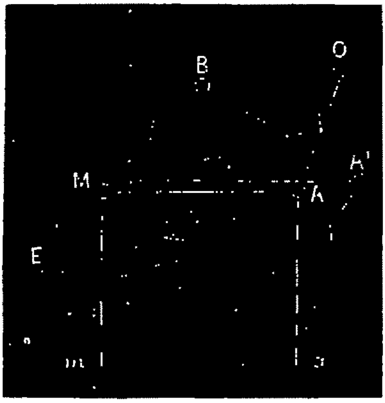

FIG. 6.

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[1] I should not have mentioned this possibility, had not a case of Westphal's (cf. Spamer, p. 541) made such an interpretation probable.

[2] The rapidity with which this occurs may seem an objection to the explanation. But the sensorial aphasia of Wernicke through lesions of A goes on diminishing likewise, a fact not susceptible of another interpretation.

with the innervation of the movement-representations the words are revived in A′ and make understanding possible; The commissure O A′ and A′ E, require to be used for a much longer time, before the disturbance in the writing can be overcome by their substitution.

The case I have just related is by no means an isolated one, but appears to be of the same category as that of Schmidt,[1] and that of Broadbent[2] mentioned by Skwortzoff as one of word-blindness. In other instances, the observations are not sufficiently full to allow us to decide whether the cases really belong to the same group.

7. The last interruption to be considered, that in the path A *a* does not really belong to the subject of aphasia, for the faculty of speech remains perfect. Still it is necessary to consider it here, because its symptoms cannot be properly understood except in connection with the present subject.

From the diagram we should conclude that there is a loss of:

(*a*) understanding of language;
(*b*) faculty of repeating;
(*c*) faculty of writing to dictation.

The following are preserved intact:

(*d*) volitional speech;
(*e*) volitional writing;
(*f*) understanding of writing;
(*g*) reading aloud;
(*h*) copying words.

There is no paraphasia nor paragraphia, because the path B M A B is whole; we could therefore designate such cases as "Isolated speech-deafness," as the incapacity for repetition and writing to dictation may be included under the term "speech deafness." But since there is a form of speech-deafness, in which, as we have seen, the faculty of repeating and writing to dictation is preserved, it is advisable to distinguish sharply these functions.

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[1] 'Allg. Zeitschrift für Psychiatrie,' 1871, vol. 27, p, 304; quoted by Kussmaul, p. 176.

[2] "Cerebral Mechanism of Thought and Speech:" 'Med. Chir. Transactions, 1872,' vol. lv.; quoted after Skwortzoff, loc. cit., p. 38.

I have a personal observation to record of such a case which has already been published by Burckhardt.[1]

Case IV.—*Chronic bronchitis. — Arterio-sclerosis. — Two apoplectic seizures; after the first, paraphasia and paragraphia gradually receding; after the second, persistent word-deafness.—Incapacity of repeating and of writing to dictation.—No disturbance of speech.*

Mr. L., aged 55, formerly a teacher and journalist, remained healthy till 1877. He then had an apoplectic fit, which was followed by troubles of language only, with the exception of a slight weakness in the muscles of the mouth on the left side of the face. These are fully described in Burckhardt's observation, to which the reader is referred for particulars. They consisted in paraphasia during volitional speech, repetition of words, and reading; paragraphia in writing to dictation. Volitional writing was very faulty, but not abolished. Nothing is said of the intelligence of language; but from the context and information given by his wife, it appears to have been intact. Evidence as to his understanding written language is wanting.

This association of symptoms may be identified with the one described under 3, as due to a break in M A, and the case is one I had in view when I said that the conditions of voluntary writing agree best with Diagram 2.

The state just described gradually improved, and a slight defect of speech only remained, consisting in the occasional misuse of a word.

In June 1882 he had a sudden attack; the symptoms seem to have been slight. A medical examination made immediately after showed an increase in the facial paralysis; and his wife said the defect of speech was more accentuated; he could not read nor write. We have here a discrepancy between the information given by the wife and that furnished by Burckhardt, who does not mention any disturbance of speech, and says he could read and write. I have mentioned the former statements because they do not agree so well with my views; but as they were made long after the event they do not deserve the same confidence as the notes taken at the time by Burckhardt. He was completely word-deaf.

The disturbance of language rapidly improved, even beyond the state in which he was before the attack. He recovered reading and writing completely; but the word-deafness remained the same. Intelligence free: patient felt weak, and did not like to leave his bed; he suffers from chronic catarrh and asthma.

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[1] "Ein Fall von Worttaubheit."—'Correspondenzblatt für Schweizer Aerzte,' 1882, No. 20.

I saw him first in July 1883; and again in June 1884; on both occasions his condition was the same.

[Passing over the general examination of the body, which presented no features of importance, we come to the functions of language.]

Understanding of speech.—Patient gives the impression of an absolutely deaf man, and differs in this respect from other cases of word-deafness. One cannot have the least communication with him except through writing; even his wife cannot make herself understood otherwise, as he can read from the lips but very few words, and these imperfectly.

I should probably have taken him for really deaf had I not been assured that he was acute of hearing, and could perceive all noises, which he said hurt him; and had I not tested the fact myself. Having written for him to raise his hand as soon as he heard the sound of a bell, he did so correctly, even when it was rung most gently behind him. It is the deficient attention he pays to sounds which gives one the impression that he is deaf. Then a few minutes later I rang again, but he did not react to the noise by raising his arm a little, until it was very loud. The same want of attention is manifest when one talks to him. He does not make the efforts to understand observed in patients with word-deafness, but remains indifferent; one has to push or shake him in order to make him attend. He hears when one whistles or sings, but does not recognize melodies. When his children sing in his room quartettes, of which he formerly was fond, he tells them to stop, and that they make too much noise. When I played the national anthem before him, he said, "Once more, I shall perhaps recognize it." But he cannot do so.

Speech.—He speaks with absolute accuracy, but with a slight drawl; very rarely, not oftener than would a healthy man, he stops for a word. He finds substantives, even complex ones, and proper names. I showed him a picture, and he said without the least hesitation, "Winterthur." On neither occasion could I detect the least trace of paraphasia.

Writing.—He writes fluently and correctly, and composes long articles for journals, of which I have seen several, and possess one.

Repeating is impossible to him, even when he is told by writing to do so. Thus when I said, "Ich heisse," he fixed his eyes upon my lips and finally brings out an "Ich," but nothing more.

Writing to dictation.—Asked to do so, he said: "But I can't hear." I nevertheless proceeded to dictate, upon which he remarked: "What a farce to dictate when one does not hear!"

Copying and reading aloud he carries out correctly and fluently.

Intelligence of written language is intact, as shown by what has already been said. He copies an I O U, written by myself, gives it to his wife, remarking, "You see you have got money."

Intelligence perfectly normal. The articles he writes are up to his usual standard and are often published in his newspaper. Advice from his medical attendant informs me that he has remained *in statu quo* up to the present date.

There is in this case only one point which needs discussion here. It is whether the word-deafness produced by the second attack really caused no disturbance of speech. The patient had preserved from his first attack a difficulty with reference to language and writing. If we follow the description of Burckhardt, the second attack brought no change into this condition, and the slight alteration of speech which remained receded completely in the subsequent evolution of the case before I saw the patient. This is in contradiction with the wife's account, who, in answer to my questions, said that the speech had been temporarily worse after the second attack, when writing and reading disappeared for awhile. I have already said that these statements were not trustworthy, having been made three years after the event, whilst the observations of Burckhardt were noted at the time; but I deemed it necessary to mention them precisely, because they seemed to militate against my view. However, even if they were correct, I would adhere to my interpretation, and assume that, in the second attack, the centre A of the diagram had been transitorily affected also. Thus Wernicke's type of aphasia, which includes, besides word-deafness, paraphasia, agraphia and alexia, might have come to be observed for a short time. This state of things would have passed away quickly, and word-deafness only persisted, complicated with a gradually receding trace of the disturbance of speech left after the first attack. This interpretation is obviously more probable than the assumption, that the disturbance of speech was a direct symptom of the focal lesion of the second attack, and was transitory only, whilst the verbal deafness remained. The opposite course is indeed the usual one, verbal deafness disappearing very much more quickly than paraphasia.

In other respects also the case offers peculiarities. I have already adverted to the possibility of looking upon the patient as slightly deaf. The other cases of word-deafness I have seen were different in this respect; one was apt in them to overlook the symptom, the patients answering all questions, but not appositely, and the danger being then to diagnose confusion of ideas. Our patient, on the other hand, paid no attention to the questions, never returned any answer, and thus gave one the impression of being deaf.

Finally, the persistence of this symptom differentiates the present case from the others. Yet these peculiarities can lend only a high degree of probability to this differentiation; a single case does not warrant certainty, and further instances are required before we can decide. The case seems to be an isolated one; I have suspected from the description of others that they belonged to the same category, but the want of accuracy in the observations has not allowed me to reach a definite conclusion.

I think I have so far shown, that each of the seven forms of aphasia postulated by the diagram are found to exist; but there remains the question, as to whether all cases hitherto observed are reducible to these forms. If we examine a large number of the cases on record, it will be found that the majority present deficiencies of observation which allow the test to be applied to them only up to a certain point. In a large number of instances the probability is that they do really belong to one of the forms. But one readily obtains examples in which this is not the case; they seem to differ in one point or another from these morbid types. Do they constitute a serious objection to my theory? I do not think so; most of them can be shown to be reducible to the schema. I must first advert to the fact, that the seven forms hitherto discussed have their origin in *simple* interruptions; whilst there is no doubt that more than one of the paths may be affected simultaneously. A decisive proof of the reality of such combination types is to be found in "total aphasia," in which there is complete incapacity to speak (the "logoplegia" of the French), with word-deafness. Here there must be a break in the centripetal as well as the centrifugal portion of the arc. We possess several observations to this effect, in which the autopsies have confirmed this assumption. Under the definition of total aphasia just given, this type will include all cases where there is loss both of speech and of understanding of speech; a combination which will arise not only when M and A are injured, but also when other combined breaks in the two branches of the arc do occur: it will be evident from the diagram that six such combinations are possible, each giving rise to the symptom. It is very doubtful, however, if these theoretical possibilities are all embodied in actual cases; their respective degree of probability can be arrived at only when we know more clearly the anatomical disposition of the nervous paths. We must assume, however, that some other combined lesions occur besides that of A and M; certain observations point to this conclusion. Thus, for instance, the celebrated case of Lordat, which, though I know only through the fragmentary account of Kussmaul, may be explained on the assumption of a simultaneous break in M B and A B. It appears that, like our patient Schwarz, he could read spelling-wise, though without understanding what he did read. Aphasia due to lesion in M B and B A, would be differentiated from that due to lesion of M and A by the preservation of the faculty of reading aloud, of repeating words, and of writing to dictation; but there is no mention made of these functions in the account just referred to.

[Whilst the present article was going through the press, I had the occasion to read the original account of the case of Lordat given by himself.[1] What I have just said has been confirmed, so far that Lordat could repeat words; but no mention is made of any trouble of writing. Certain statements, however, make it doubtful as to whether he was suffering from aphasia, and not from paraphasia only. He says: "There was not only amnesia, but also what I would call paramnesia, that is, an erroneous use of known and remembered sounds. Thus when I wanted a book, I asked for a pocket-handkerchief. True that immediately after having pronounced this word, I withdrew it; I felt that another was wanted. Another mode of paramnesia consisted in interverting the letters of the syllables in a compound word, of which I had recovered possession. Thus for 'raisin' I said 'sairin'; for 'musulman,' I felt inclined to say 'sumulman.'"

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[1] Grasset, "Contribution clinique à l'étude des aphasies."—'Montpellier Médical,' Janvier 1884.

These data are somewhat opposed to those which precede them, and which leave no doubt that, with the exception of a few words, Lordat was quite speechless. I am therefore disposed to assume, that this paraphasia appeared only later, in the period of regression. The explanation given above would thus be perfectly justified; for I have already shown, that paraphasia and amnesia are observed during the healing stage of aphasic lesions in B M.

This supposition is the more plausible, that the quotation from Lordat shows that the word-deafness had already left him; he was conscious of the mistakes he made in speaking.

If the case of Lordat had been from the very beginning one of paraphasia and not of aphasia, it would agree in the main with that of our patient Schwarz (Case I.); and we should explain it simply on the assumption of a break in the path A B.]

Besides such combinations, we may conceive others due to the simultaneous implication of paths on the same side of the arc. The types thus obtained would coincide, to a great extent, with those arising from single breaks; the one due to interruptions in M A and A *a*, however, would present a peculiar clinical picture, distinguished from Wernicke's sensorial aphasia, inasmuch as the understanding of writing would be preserved. I have found no case illustrating this form. Wernicke, however, in his explanation of his type of aphasia, expressly specifies that the faculty of reading may be intact, but the reasons he gives for this fact are quite different from mine.

Further examples of combination may arise from the implication of the nerve-paths interested in the production of written language only, A *o*, A E, and O E (Fig. 4). It is probable that the path O E, at least, is sometimes involved, for there are cases in which it is expressly stated that the power of copying was affected, as for instance in that of M. Valentin.[1] To judge from the symptoms, this must have been a case of total aphasia caused by a lesion of M and A, with a break in O E.

It is not necessary for me to go through all the possible combinations; many of them are very unlikely to occur in reality.

A second, and very important circumstance which must be kept in view when we deal with cases which appear to militate against our views, is that the several components of the various types disappear at very different rates. A large number of "aphasics" fall under our notice at a variable period after the attack has taken place, when some of the symptoms have already begun to disappear. It is easy to show, by examples, how even a few days may be enough to efface some of the characteristic symptoms of the type; this is what happened, for instance, in our third Case.

We must always bear this fact in mind, when in presence of complicated morbid types of unrecent date. Our task is obviously made more difficult, though we must not despair of elucidating the problem; for there is no doubt that there is a law governing the order of retrogression of the various symptoms, at least when they are not obscured by the interference of indirect focal symptoms. These laws are to be derived from a sufficient number of exact observations. To our small stock of knowledge on this point belongs the fact of the usually rapid disappearance of word-deafness, and the greater persistence of the troubles connected with written language. This fact appears to me to throw light upon a not unfrequent type which does not readily fall in with our classification; I mean that of motor aphasia in which alexia[2] co-exists.

A break in the motor tract can never in itself produce a disturbance in the intelligence of writing; and according to the schema there must then be a lesion of the path A O also. Such a combination, exclusive of the simultaneous implication of *a* A B, is obviously not impossible, but, as we shall presently see, all our actual notions concerning the localisation of aphasic phenomena compel us to admit that it must be a purely accidental one. Now the type I am referring to is so common, according to my own experience, and as shown in Trousseau's *Clinique Médicale*, that we can scarcely admit of an accidental occurrence. An explanation must therefore be found for what would otherwise be a serious objection to the theory proposed in this article.

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[1] 'Revue Médicale de l'Est,' xii. 6. Quoted by Skwortzoff, p. 63.

[2] By "Alexia" is meant here, not only the impossibility of reading aloud, but also the deficient understanding of written signs.

These cases appear to me to have been instances of total aphasia in which the symptom of word-deafness has already been recovered from; I have actually observed this course of events in a case where the initial total aphasia evolved into the type we are now considering. I have already referred to this case, of which I unhappily do not possess the notes. In other instances a careful investigation has led me to discover remains of word-deafness, consisting in a deficiency manifested by the patient when given more complicated orders to carry out, and corresponding exactly to the similar imperfections noted in the later stages of our Case III. Here is an example of the kind.

Case V.—*Aphasia appearing during typhoid fever. Doubtful traumatic cause.*

S. T., aged 49, a cabinet-maker, was admitted into the Insel hospital on the 24th of January, 1884. It is almost impossible to hold communications with the patient, owing to the disturbance of his faculty of language. All that could be made out of his previous history, was that he fell ill with typhoid during a great epidemic at Meyringen. One day he fell out of bed upon his head, he remained several weeks (?) unconscious, and was speechless when he came to himself again.

Patient is a fairly strong man; his intelligence appears to be perfectly normal; he tries with a certain degree of success to express himself by gestures. He occupies himself in the wards with the general service and with woodwork. He has made with skill several boxes, and plays dominoes and cards very well. The nurse, however, thinks he is deficient in some ways, because he has failed to carry out certain directions.

Speech.—He is absolutely speechless, with the exception of a few words such as, "yes," "no," "now," "but," "too," "just;" and the sound "gook," which he substitutes on every possible occasion.

Now and then he comes out with a longer exclamation, such as "Herr Jesu Gott," or "Min Gott im Himmel."[1]

Repeating.—He cannot repeat anything said to him except "yes" and "no."

Intelligence of speech.—On a superficial examination the patient appears to understand everything. He points to the right objects when they are named, and carries out simple orders perfectly, shows his tongue, closes his eyes, stands on a chair, shuts the stove, &c. But when the directions are more complicated it becomes evident that he does not understand so well. When asked, for instance, to bring a large bottle standing near a basin, he takes the basin. If simple but unusual things are told him, he does not do them correctly. I asked him one day, on coming near his bed, to show me his nose, and he pulled out his tongue. Suggestive questions are rightly understood by the patient.

Intelligence of writing.—He formerly read and wrote fluently, but now never takes up a book or newspaper, and manifests on careful examination a great loss in his power to understand written language. He apparently manages a few words; for instance, "soldier," "sheep," "horse," "pigeon." He signifies by gestures that he understands the words "wine," "Albert" (the name of his son); short sentences likewise, such as "eyes shut," "mouth open." But beyond this he is incapable of carrying out plain directions written in large characters, though he points to the letters as if to indicate that he knows them. He has great difficulty, however, in picking them out of a box, and seems to have no idea of their alphabetical order. His difficulty in understanding writing is greater than that in recognising spoken words, though the disturbance is obviously analogous in both cases.

Writing.—The patient writes his name correctly, but he cannot write anything else.

To *Dictation* he makes illegible scrawls, except for his own name, which he writes correctly.

Copying.—He copies everything, but in a servile manner, and with all the mistakes made in the model.

Reading aloud absolutely impossible. He knows the numbers, can write them also, even when with two digits. He often makes mistakes in the multiplication table.

In other respects patient is quite well. No abnormality discoverable about the head; all functions depending upon cerebral and spinal innervation intact, excepting a weakness of the right hand, which gives 36 on the dynamometer whilst the left gives 55. Patient indicates by signs that this has been the case since his fall. No such difference exists in the lower extremities.

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[1] We tried to determine whether the word-representations could be excited from the higher centre. Objects were shown to him, but he could not give the number of syllables of their names, nor indicate it by pressure of the hand, according to the plan previously described. He did it correctly if the name was given to him, so that he must have understood what he was told to do.

We have to do here with an apparent case of Broca's aphasia in which the behaviour of the patient led one to suppose that he could not read at all; on closer examination a considerable diminution of the intelligence of written language was shown to exist. The understanding of spoken language appeared at first sight undisturbed, but on investigation defects appeared which were obviously remains of a transitory word-deafness; so that the case must be considered as one of total aphasia.

I have observed another case, quite analogous to this, with reference to the troubles in the functions of language; and I believe, as has been already mentioned, that Trousseau's cases must be interpreted in the same way. The inability of the patients to read was here assumed on the evidence of their not taking to books though their intelligence was good. Though they sometimes thought they could read, they persisted in ever using the same volume or the same page; similar phenomena were observed in my two patients. Trousseau, however, does not adduce any more thorough investigation of their intelligence of writing or speech.

If we take into consideration the facts, that the individual disturbances in the functions of language may occur more or less incomplete or partial[1]; and that more or less considerable remains of them may persist and thus complicate the task of elucidating the type to which a case belongs, it will not appear strange that many instances are difficult or impossible to explain. But this forms no insuperable objection to the theory. On the other hand, our hypothesis would be gravely compromised if a whole category of common aphasic disturbances could not find room within its boundaries; and thus it becomes necessary for us to consider what place the "amnesic aphasia" of authors must occupy.

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[1] I abstain from entering here into details about such partial disturbances, so as not to complicate a subject already complex enough. Their study must, however, elucidate many points connected with the mechanism of language. A remarkable start in this direction has been made by Broadbent ('Medico-Chir. Trans.' June 1884).

The first who distinguished between aphasia from loss of memory of words and aphasia from disordered co-ordination of word-movements, or disturbed conduction of volitional impulses, was James Russel.[1] Most English writers, and in Germany Biermer[2] and Kussmaul, have taken this view. For the first form Ogle[3] preserved the name of amnemonic, for the second that of *ataxic* aphasia; but the word *amnesic* has since displaced the former. Our task is to throw light upon the distinctive clinical symptoms by which these two forms of aphasia can be recognised.

Russel gave as a characteristic test that patients with preserved memory of words, but disturbed conduction of volitional impulses, still make notable efforts at speaking. According to this criterion a great number of cases which are now denoted as of amnesic aphasia would not have been considered as such by him, for the patients try hard to speak, and often possess a goodly vocabulary. Nor can I believe that efforts to talk by aphasics who have but a few words left, justify the conclusion that they have the sound-representations of others at their command. Moreover, I think I have shown that in Case V., which belongs to this category, these representations were missing.

Ogle has described as a pathognomonic sign of his amnemonic aphasia that patients can name nothing, or next to nothing, but can usually repeat words spoken before them. On this assumption, the fourth form described by us as depending upon an interruption of the path B M, would rank as amnesic. But it is certain that this definition does not exhaust all that is understood by amnesic aphasia. Most cases of it must receive another explanation.

Let us consider how, from our point of view, an aphasia due to obliteration of the memory of words is distinguished from one due to disturbance in the motor apparatus. As stated above,

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[1] 'British Medical Journal,' 1864. Quoted after Spamer.

[2] 'Correspondenzblatt für Schweizerärzte, I. 1871.

[3] "Aphasia and Agraphia."—'St. George's Hospital Reports,' ii. 83–121.—See Virchow's 'Jahresbericht,' 1867, p. 40.

I think that in most motor aphasias—in those, among others, of the true Broca's type—the patient has lost the auditory word-representations, that is to say, cannot awake them voluntarily by an action of their higher centres. The only criterion of the fact is the preservation of the faculty of writing; or else the power of putting together words from letters; or of counting syllables, their number being expressed by signs, or pressure of the hand. These actions are possible to but a small number of the patients, who are described as ataxic aphasics. Kussmaul[1] also considers the power of writing as a sign of the possession of auditory word-images; but as most ataxic aphasics have lost it, he assumed in their case the presence of an ataxic agraphia, that is, of a lesion of the motor-writing tract. I have already shown why I cannot adopt this view; nor can I accept the criterion by which the same author pretends to recognise the faculty of exciting sound-representations in themselves by those ataxic aphasics who have lost the faculty of written language. He says: "The best, but not the only proof of the existence of their faculty is given us by written language. We must assume its existence where aphasics who have never learnt to write, or have lost the faculty of writing, use expressive signs to manifest their thoughts and to answer our questions correctly. Fallacies are obviously dangerous here, and accurate investigations must repeatedly be made in order to ascertain whether the patient actually understands. No signs especially must be made to accompany what we say."

I believe that we must distinguish sharply between the power of resuscitating sound-representations of words voluntarily (i.e. from the concept centres), and the faculty of doing so acoustically (i.e. from the ear), and putting them in communication with the concept-centres. The former involves efferent, or motor, actions; the latter afferent, or sensorial, effects; involving the paths of the diagram B M A and *a* A B respectively. The test of Kussmaul has reference only to the understanding of words (i.e. to the state of the path *a* A B); and which may be lost without destruction of the auditory word-representations, as is shown by Case IV., where we find absolute word-deafness with no disturbance of writing. I have also found, that though a patient understood speech perfectly, he might have lost every power of voluntarily bringing back the word-sounds (Case II.). In most ataxic aphasias, therefore, I consider that there is, besides the impossibility of expressing any words, a loss of word-memory, that is of the power of sound-revivifications.[1] If we turn to Ogle's amnemonic aphasia, where the faculty of repeating is possible, we find that the power of speech proper is gone.

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[1] Loc. cit. pp. 157 and 158.

Loss of voluntary innervation of the auditory word-centre, but not of the motor word-centre, would occur in cases of lesion of the centre A itself, or of the commissure A M. The corresponding types (sensorial and commissural aphasias of Wernicke) described under 2 and 3, do manifest the disturbance of language which has been called amnesia or amnestic aphasia. This trouble we have, like Wernicke, associated with a lesion of the auditory word-representations; and, as the reader will remember, we find in recent cases that the vocabulary is rich (as one would expect when the path B M *m* is intact), but that speech is incorrect, the words used being mutilated or inverted. This symptom we name, after Kussmaul, paraphasia; when it is disappearing, or is but slightly developed, there occur evidences of amnesia, that is more easy to demonstrate when the patient is made to name objects than when he is engaged in ordinary talk; names which occur without effort in fluent speaking, arrest him when he has to find them for objects or persons shown to him. If the path M A be interrupted whilst A remains intact, the auditory word-representations may be called up through the auditory, as well as through the optic nerve; and it is enough to say or show the word to restore it, either for an instant or for a longer period.

These phenomena may be brought into harmony with our schematic representation, by assuming, that for fluent talking the innervation of the auditory word-centre is not necessary, the latter being connected to the path B M *m* only.

The process of finding the name of persons and objects is different. It is easy to convince ourselves by self-observation that we first seek the auditory representation of the word, from which starts the excitation for the mechanism of articulation; we are not conscious of such a process in fluent speaking. Experience further shows that the same word-amnesia occurs in the type which we have seen to depend upon a break in the path A B (see Case III.). In explanation we must assume that the nervous current, starting from the auditory representation in A, must pass through B to reach M.

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[1] Ogle admits that in ataxic aphasia, the memory for words may fail, as well as the power to express them.

The reader may find this very improbable and too complicated. But it would appear as if, in naming objects, the auditory representations once found had to react in consciousness. This variety of language is a much more "conscious" one than fluent speaking, in which we are aware of the sense of what we are saying, rather than of every word we say. Under certain circumstances conversational language is carried on in a similar way to naming, as, for instance, when we use an idiom not quite familiar to us. Here we must seek the words by the complicated process just mentioned; the direct communication between concept and motor centre without cooperation of sound-representation does not exist; the subconscious act of speaking is not yet possible. A greater psychical exertion is obviously required, and consequently more fatigue is entailed.

It seems to me questionable, however, to place amnesia on a par with the other phenomena of aphasic disturbance. At any rate, it is a pretty constant accompaniment of the types above mentioned, and of the regressive forms of motor aphasia which pass through a stage where amnesia is observed, as has been shown in the second of the cases we have adduced and in other analogous ones. But it is not a sign of a focal lesion, and appears in more diffuse morbid processes, or where the cerebral circulation is deficient, or as an early sign of the natural involution of the brain.[1] Its occurrence may be explained on the assumption, that the resistance offered by the complicated nervous path can be overcome only when the latter is in a normal condition, and when the functions of the cerebrum are fully carried out. Thus amnesia may be found as a residuum after lesions in B M A B, and appear as a symptom of diminished cerebral activity. At any rate, it cannot be accepted as a basis for the classification of aphasias, and the word "amnesic" aphasia had better be abandoned. The expression "ataxic aphasia" is equally unsatisfactory; we have not to do with disturbed co-ordination of word-movements.[1] The misnomer has probably originated in the fact, that movements other than those of speech can be performed by the patient with his vocal organs; but from our point of view, aphasic speechlessness is more of a *paralytic* nature.

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[1] The various forms of amnesia, the senile as well as those observed in aphasic disturbances, do not offer essential differences; they always involve proper names chiefly, then substantives; very rarely verbs, adjectives and pronouns. Kussmaul explains this fact thus: "The more concrete the idea, the more readily the word which expresses it escapes the memory when it is failing. The reason of it is that the representations of persons and things are more loosely connected with their names than the abstractions of their conditions, relations

We have thus reached the subject of nomenclature, and should advert to the best way of designating the seven simple forms of aphasia we have described. The best plan is to adhere, as far as possible, to the names already accepted and in use. In a preliminary notice I gave last June, I followed the division of Wernicke, and designated aphasia from breaks in the efferent path B M *m* as *motorial*, and that from break in the afferent B A *a* as *sensorial.*

Further consideration led me to a different result. As long as one thought, with Wernicke, that disturbances in the understanding of language were always associated with troubles of speech, the former would be called "aphasias"; but this is not the case when we deal with morbid types in which speech is perfectly intact, such as that described under No. 7. On the other hand, it is obviously necessary to bring together lesions

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and properties. We represent persons and things to ourselves as easily without as with their names; the sensory representation (Sinnenbild) is more important here than the idea (Sinnbild), the name contributing but little to the knowledge of the person or of the object. Abstract ideas, on the other hand, necessitate the aid of words, which form their sole embodiment. Hence verbs, adjectives, pronouns, and still more adverbs, prepositions and conjunctions, are far more intimately associated with thought. We may imagine that there is a much greater number and combination of excitations of the cerebral cells required for the genesis of an abstract than of a concrete idea; and that there are in consequence far more numerous organic connections between the former and their corresponding word-forming process, than in the case of the latter."

[1] As Sander has already shown. 'Archiv für Psychiatrie,' II. p. 40.

in B A *a* under one appellation. Hence it seems to me more correct to accept the nomenclature of Kussmaul, and group together those affections, designating them as *word-* or *speech-deafness.* The imperfection in the understanding of language is, of course, but a partial symptom in this type; but the same is the case for the speechlessness, or aphasia, symptomatic of a break in B M *m*, and word-deafness bears to the symptoms of a lesion in B A *a*, the same relations as aphasia to those of a lesion in B M *m*. It constitutes the dominating and necessary sign thereof, around which the others are grouped, just as agraphia and the other disturbances centre round the aphasia, in lesions of B M *m*.

We shall therefore do well to reserve the name *aphasia* for the latter class; and, as just mentioned, designate the former by the term *speech-deafness*, or *logokophosis.* Each of these groups fall into three subdivisions: lesions of the centres (M and A in the diagram), might best be said to give rise to *central* aphasia or word-deafness; whilst the others would be distinguished as (*inner* and *outer*) *commissural* aphasia or word-deafness. The disturbance arising from a lesion in M A could provisionally receive the name of *commissural paraphasia,* until its localisation be fixed with certainty in the Island of Reil, when it would be called *insular aphasia.*

I am well aware of the objections which may be raised against this nomenclature. It would have been more rational not to give the name of a prominent symptom to the leading morbid types, and to find new names for them; but I shrank from an attempt involving the substitution of new names for old and familiar ones, and feared only to create more confusion thereby.

Some readers will object, that the nomenclature rests upon a still hypothetical basis; but, in my opinion, it is of assistance to make it rest upon the assumptions involved in the explanation of the problem. Should this explanation, perchance, prove to be erroneous, no harm will have resulted thereby, and we shall have only to alter the names.

We cannot conclude without having previously considered how far we can proceed with the cerebral *localisations* of the various forms of aphasia. We must obviously start from those data which may be considered as most certain and best defined. The origin of Broca's aphasia in disease of the inferior frontal convolution will scarcely meet with any doubt, and Wernicke's explanation of his sensorial aphasia by a lesion of the neighbouring temporal convolution has been confirmed by subsequent experience. We may thus assume that the points M and A of our diagram occupy these parts respectively. The path M A has been referred by Wernicke, on anatomical grounds, to the region of the insula; and it is of common occurrence to see lesions of this spot cause disturbances of speech; but we cannot as yet affirm that the latter corresponds to our type (through lesion of A M), though Case II. gives support to this view. There we see the clinical association of symptoms accounted for by the destruction of the insula; but the lesion extended to the neighbouring frontal and temporal convolutions, and no data could be obtained concerning the severity of the apoplectic seizure. It is, however, probable that the trouble of speech was an immediate effect of the lesion, because it persisted unaltered more than a month, till death supervened, whilst the other symptoms underwent a more or less considerable retrogression. A case[1] of Meynert's, though somewhat fragmentary, might be adduced here also.

We shall further have to deal with the course of the inner and outer commissures of M and A. Though in the diagram B is represented as a sort of centre for the elaboration of concepts, this has been done for simplicity's sake; with most writers, I do not consider the function to be localised in one spot of the brain, but rather to result from the combined action of the whole sensorial sphere. Hence the point B should be distributed over many spots; and the commissures M B and A B, would not form two distinct and separate paths, but consist of converging radiations from various parts of the cortex to the points A and M (see Diagram 7, p. 478). This admission does not do away with the possibility of interruptions in the commissures B M, B^1 M, B^2 M, &c.; but leads us to expect, that any simultaneous break in them must occur close to their entrance into the lower centres M and A.

Hence we shall have to look for the lesion which produces inner-commissural aphasia in the white matter of the hemispheres, namely at the base of the third frontal convolution. The anatomical localisation of inner-commissural word-deafness will likewise have to be sought near the first temporal convolution. We have already had to observe more than once,

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[1] 'Wiener medicinische Jahrbücher,' 1876.

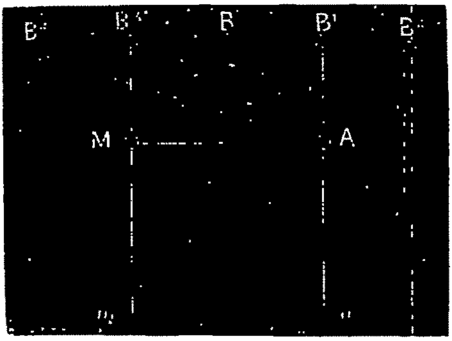

FIG. 7.

that in most published cases, the description of the symptoms is deficient; their morbid anatomy likewise leaves room for many doubts.[1] But though we are still in want of demonstrative evidence, we occasionally find indications in support of our views. Thus in a case of Farge,[2] we find that the patient had right hemiplegia after a severe apoplectic seizure. He could say only a very few words; but could repeat correctly what was said, at least the first words of a sentence. He died, as will be seen in a reference by Nothnagel,[3] twenty days after the fit. The autopsy revealed integrity of Broca's convolution; but in the white matter beneath was found a patch of softening, the size of a small egg.

There is no other case, besides my own, of inner-commissural word-deafness published in sufficient detail to allow of a certain diagnosis. The one by Broadbent[1] furnished an autopsy, but as it came under observation several months after the attack, and as a portion of the symptoms had certainly retrograded by that time, there is some difficulty in identifying the type. There were slight word-deafness and paraphasia present; understanding of writing was highly deficient. The patient could write to dictation, copy and repeat words; he died a month afterwards from another attack, when an extensive hæmorrhage took place in the left hemisphere and broke into the lateral ventricle. This fresh lesion made it difficult to recognise the older one; but it appears that the left temporal convolution bordering the Sylvian fissure was intact. A small hæmorrhage was found in the lowest temporal convolution. The focus, the appearance of which made Broadbent connect it with the symptoms, was in the white matter of the temporal lobe, between the posterior end of the Sylvian fissure and the lateral ventricle near the root of the cornu ammonis. The case proves nothing positively, and all which may be said is that it rather harmonises with our hypothesis.

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[1] In Hammond's 'Diseases of the Nervous System,' there is a clear case of aphasia, without loss of power to repeat words, quickly followed by death. The case was one of injury, and then were found (1) an ecchymosis the size of ½ dollar in the right frontal lobe; (2) a tear of the right middle meningeal artery, with large hæmatoma. It is obvious that such an observation cannot be utilised for our purposes.

[2] 'Gazette hebdomadaire, 1865,' 44. See Kussmaul, p. 93.

[3] 'Topische Diagnostik,' p. 358.

The difficulties are still greater when we turn to the localisation of the paths M *m* and A *a*. We know the terminal points of the former; the third frontal (Broca's) convolution, and the motor nuclei of the medulla oblongata, which enter into activity during the act of speaking. But we know practically nothing of the actual course of this commissure, whether it is bilateral, or runs exclusively between the left hemisphere and the basal organs. The usual view is that the bilateral movements of the lips, tongue, palate and larynx, which enter into play during speech, may be excited from each hemisphere; but that the connection between this process of innervation, and the intelligent concepts and auditory word-representations necessary to the combination of those movements into articulate speech, occurs on the left side only. It thus comes to pass that only the left third frontal and its efferent fibres are engaged in the movements of speech, whilst the corresponding organs on the right side govern the movements of the organs of speech, but have no share in co-ordinating them into the expression of words. The consequence is that a lesion on the left side gives rise to a disturbance of speech without apparent loss of motility, whilst a bilateral lesion is required to produce a paralysis of the motor organs.

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[1] "Cerebral Mechanism of Thought and Speech," in 'Med. Chir. Trans.;' quoted after Skwortzoff, p. 88. Virchow's 'Jahresbericht,' 1872, II. 45.

Now if the fibres from Broca's centre reached the basal organs down the left side only, the usual persistent aphasic symptoms would arise from lesions of the left peduncle or internal capsule as uniformly as they do from those of the centre itself. But we know that this is only exceptionally the case; hence there must be a partial decussation of the speech-tract from left to right hemisphere within the brain itself, so that the left internal capsule does not contain the whole bundle of those fibres. Diagram 8 is intended to represent this fact, and shows why the usual lesions of the left internal capsule are not followed with permanent troubles of speech. We see that both hemispheres partake in the function of language,[1] and that bilateral disease is necessary to set up disturbance of speech, combined with deficient movement of the tongue, lips, &c.; such cases, however, are no longer looked upon as instances of

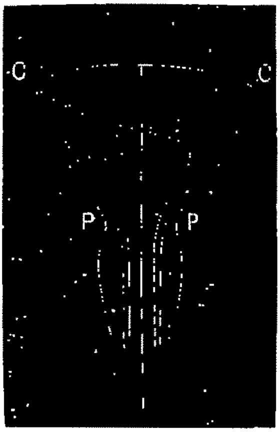

FIG. 8.

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[1] This function is more frequently interfered with by lesions of the deeper portions of the *left* hemisphere, a fact which may be explained on the assumption, that the direct fasciculus is more considerable than the crossed one. The great individual differences observed with reference to this point may rest on corresponding divergences in the relative distribution of fibres in these fasciculi.

aphasia, but have been classified under the name of "Pseudo-bulbar paralysis."

I do not think Wernicke justified when in his last paper[1] he includes under aphasia the speechlessness occurring in the terminal period of progressive bulbar paralysis through total destruction of the motor nuclei. The term aphasia should, I think, be strictly limited to those cases of speechlessness which are unaccompanied with any disturbance of the mechanism of articulation, though I am aware of the difficulty of always accurately defining aphasia from anarthria. When an aphasic is deprived by a fresh right-sided hæmorrhage of the movements of the tongue, lips, &c., we are enabled to recognise the aphasia among the symptoms of pseudo-bulbar paralysis only by our knowledge of how the affection was developed. But occasional difficulties of this sort cannot make me abandon the secure ground afforded by the discrimination between aphasic and anarthric phenomena.

On the assumption that a portion of the fibres decussate, as just suggested, in the hemispheres themselves, an explanation is afforded of all the phenomena which led Wernicke to assume that the right cortex has a share in the production of speech. I hold that no other fact referring to this function is more solidly established than that of the origin of the speech-tract from the left side only. We are not justified in attacking it, as long as we can reconcile with it the data of experience. Now all the facts adduced by Wernicke harmonise perfectly with my view; but whilst his explanation does not account for the regular occurrence of aphasia in disease of Broca's convolution and its non-occurrence in lesions of the left peduncle and internal capsule, these phenomena are the natural consequences of my hypothesis.

Lesions of the deeper portions of the brain, followed by disturbance of speech, moreover, do not bring about aphasia with its characteristics as a "verbal" trouble; the formation of sounds is more or less prominently interfered with. This verbal characteristic of the cortical symptom is a natural consequence of the functions now usually attributed to the cerebral centres in general, and of the innervation of those concerned in language especially. If we assume that Broca's sphere is the seat of acquired motor word-representations (kinæsthetic centre), and that its lesions obliterate these memories, and so give rise to aphasia, it follows that its destruction must affect *words*, and not *sounds*; for those representations acquired during childhood are associated with words only. It is at a much later period of development that we have learnt to decompose words into sounds. As the elements for the formation of words are deposited in the cortical centres of speech, so the dispositions for the production of sounds are made in the bulbar nuclei. The paths, therefore, between Broca's centre and the nuclei will have to undergo a corresponding conversion. The question is now where this conversion takes place; whether low down in the pons, as Wernicke supposes; or, as I think, much higher up, basing my opinion on the fact that disturbances of speech, arising from lesions of the deeper cerebral regions, do not, as just mentioned, partake of a purely "verbal" character.

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[1] "Ueber die motorische Sprachbahn und das Verhältniss der Aphasie zur Anarthrie."—'Fortschritte der Medicin,' 1884.

All these considerations compel us to assume, that only a short extent of the efferent tract from Broca's centre is so constructed as to give rise, on being injured, to real aphasic disturbances. We shall therefore have to look also for the lesion of aphasia without agraphia in the white matter of the hemispheres, though we can as yet scarcely conjecture its localisation. Two new contributions,[1] the results of which, however, are scarcely concordant, point not to a simple radiation of the fibres from the Broca's convolution towards the internal capsule, but to a more complicated arrangement.

The course of the outer commissure A *a* is still more problematical, though one may, in a certain measure, circumscribe the region which it must occupy.

Both ears are capable of receiving and transmitting excitations of speech, but intelligence of language is bound up with the activity of the left hemisphere only; so both acoustic nerves must enter into relation with the latter.[1]

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[1] Wernicke, 'Fortschritte der Medicin,' 1884. No. 1. Bitot, "Du siège et de la direction des irradiations capsulaires chargées de transmettre la parole." —'Archives de Neurologie,' 1884, 22, &c.

The affection due to a lesion in A *a*, characterised by word-deafness, can obviously come into existence only if the irradiations of both acoustic nerves in the left temporal region are broken through.

With reference to the point where the auditory tracts combine to form the path A *a*, we might place it low down in the bulb or cerebellum, and assume that the fibres ascend already, distributed as they are in the cerebrum itself. Such a view would be opposed to the clinical evidence we possess; there are numerous cases of hemianæsthesia through lesion of the posterior part of the internal capsule, in which a diminution of hearing of the affected side existed. Hence the union of the two tracts does not seem to be effected below this point. A unique observation by Vetter[2] of complete deafness of one ear with hemianæsthesia on the same side, of cerebral origin, lends further support to this view. The union, therefore, must take place in the cerebral white matter, and we shall have to look for the localisation of the path A *a* and its lesions in this region, and probably in the temporal lobe.

It is obvious that lesions of the cerebral white matter may not always give rise to the associations of symptoms we have described. Apart from indirect lesions of the cortical centres, complex interruptions in the commissures M B and M *m* on the one hand, A B and A *a* on the other, will give rise to clinical types corresponding exactly to those of Broca and Wernicke.

I have still to advert to the phenomena of *word-blindness*. As we have already seen, this symptom arises in common with others from breaks in different tracts subservient to the function of language. If we were to give to it the same extension as does Skwortzoff, for instance, we obviously could not speak of its being uniformly localised. This condition might rather be fulfilled in cases of isolated word-blindness. Here we have not to do with a simple obliteration of the visual representations of the written signs, with a lesion of the point O in the diagram. These patients can copy, hence the path O E must be intact; and from what we know of the cortical distribution of the optic tract in man, it is certain that the visual memories of words must be stored up in bilateral spheres. Isolated word-blindness is, as has been said by other observers, the result of a break between the visual and the auditory centre of word-representations; that is to say, of a break in the path O A after the union of the converging tracts from both occipital lobes. The observations bearing on the point are few, but they support this conclusion; the anatomical subtratum of word-blindness has been found to be a focal lesion in the neighbourhood of the left inferior temporal lobe.

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[1] Since a similar distribution must obtain in the right hemisphere, crossed deafness would not be produced in man by destruction of one auditory sphere (as Munk says is the case for dogs). In fact there is no record of observations showing left deafness to be due to a lesion of the right hemisphere.

[2] "Ueber die sensoriellen Funktionen des Grosshirns;" 'Deutsches Archiv für klin. Medicin,' xxxii. p. 498.

Though I have ventured the above remarks concerning the probable localisations of aphasic disturbances, I am well aware of the restricted foundations on which we may safely build, and what a space theoretical reasoning has still to occupy in the discussion. If I have, nevertheless, not kept my views to myself, it was on the principle, that we must not recoil from the consequences deducible from our hypotheses. In proportion as we draw these conclusions, we shall obtain the necessary data whereby to correct, or if need be abandon, them. Again, no simple observer can collect material enough to accomplish this task, which requires the co-operation of many; and under these conditions even erroneous assumptions may prove of advantage in the search for truth.

ON THE NERVOUS LESIONS PRODUCED BY LEAD-POISONING.

(*From the Pathological Laboratory of the Owens College.*)

BY ARTHUR ROBINSON, M.B., C.M.

THOUGH cases of lead-poisoning are of frequent occurrence, up to the year 1880, when Dr. Moritz[1] published his paper on Lead Palsy, only seventeen cases had been placed on record in which a satisfactory post-mortem search for the cause of the paralysis had been made; Dr. Moritz's case brought the number up to eighteen.[2] Since 1880 two other cases have been published. One by Oeller,[3] who found the following changes in the nerves and spinal cord of a man, aged 40, who had worked in lead for a year and three months, and who had suffered from paralysis of the extensor muscles of the wrist and fingers of the interossei of the hand, and of the muscles of the thenar eminence.

Spinal Cord.—Small hæmorrhages were found in the middle of the anterior horns, and at the junction of the anterior and posterior horns, in the anterior longitudinal fissure round the large blood-vessels, and also in the white substance of the cord. In the cervical enlargement, from the fifth to the eighth cervical nerves, small spots of softening were found. Spots of softening were also found in the middle of the anterior horns and in the anterior part of the posterior horns. In these positions the ganglion cells were small, atrophied, highly-pigmented, homogeneous, hyaline, and devoid of processes;

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[1] 'Journal of Anatomy and Physiology,' 1880.

[2] Lancereaux, 3; Gombault, 1; Westphal, 1; Tiburtius, 1; Friedländer, 1; Ollivier, 1; Zunker, 1; Eisenlohr, 1; Vulpian, 1; Déjérine, 5; Monakow, 1; Moritz, 1.

[3] Oeller, Thesis, Munich, 1883.

they stained badly, and no nuclei were visible in them. Others of the ganglion cells contained large vacuoles. There was a number of spider cells.

The white substance of the cord was only affected in a subordinate degree. Some of the fibres of the musculo-spiral nerve were atrophied, and there were the usual changes in the muscle. Oeller concludes, that the central changes were the primary and essential part, and that the hæmorrhages, following on the general arterial degeneration, were the cause of the softening.

The second case is recorded by Duplaix.[1] The patient was a man, aged 47. He had been a painter for many years, and had been very feeble for a year. There was no trace of syphilis or alcoholism.

The patient noticed, when the feebleness first affected him, that his limbs rapidly lost flesh, and finally the emaciation extended to his face and trunk. The muscular feebleness increased, and at last he was unable to leave his bed. Sensation was normal. He was affected with purpura, œdema of the lungs set in, and death rapidly followed. The post-mortem showed granular kidneys, a small heart, and atrophied muscles.

The spinal cord and the nerves were carefully examined, marked changes being found in both.

Spinal Cord.—The neuroglia was increased in amount, forming bands of tissue enclosing spaces filled with nerve-tubes,

The nerve-tubes were also separated by fine fibrils of connective tissue and many round nuclei. The vessels throughout the whole cord were sclerosed and surrounded by a dense connective tissue.

The nerve-cells in the anterior and posterior horns were unchanged. In the anterior roots, distinct changes were discovered in the structure of the nerve-fibres. The alteration did not extend over the whole length of a fibre, the affected portion being continuous at either end, with an absolutely healthy portion.

The disease usually extended over three or four internodal

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[1] Duplaix, 'Arch. Gén. de Méd. 1883,' vol. 2.

segments. Rarely was one segment affected alone. In fact, the changes were those described by M. Gombault, under the name "phase of restitution."

The myelin sheath was everywhere present, but some portions of the fibres were thinner than others, the diminution in volume affecting both the axis-cylinder and the myelin sheath. Nerve-fibres were also seen exhibiting swelling of the white sheath, and also of the axis-cylinder, and in some places there were empty tubes, the nuclei of which had proliferated. The posterior roots were healthy.

The nerves showed both degeneration and restitution. The muscular fibres had atrophied, and their nuclei had proliferated.

The following case was admitted into the Manchester Royal Infirmary, under the care of Dr. Leech, whom I have to thank for kindly permitting me to make use of it, in December 1882. The patient, J. C., a man, aged 30, had been a painter for fourteen years, but for three years before his admission he had nominally been a cabinet-maker, though he still occasionally did a little painting.

He had his first attack of "drop wrist" in the summer of 1877; it continued through the winter, but he recovered in the following spring. Every winter after his first illness, the attack returned and laid him up until the spring.

He was troubled with frequent micturition, but had never had colic or cramps.

The muscles of both forearms were very much atrophied, the extensors of the wrists and fingers were completely paralysed, while the flexors retained a fair amount of power. The muscles of the thenar and hypothenar eminences were also atrophied, and the patient could not oppose his thumbs. He could pronate, but could only partially supinate both forearms. The peroneus brevis and longus muscles on the left side were atrophied and paralysed, and those of the right side were affected with paresis. All the atrophied muscles showed the reaction of degeneration when tested with electricity.

The patient was treated with iodide of potassium, and the interrupted and continuous currents were applied to the affected muscles. He gradually improved, the atrophied muscles increased in size, and to a certain extent regained their power. He was dismissed in March, 1883, very much improved.

He was readmitted in June 1884, suffering from œdema of the lower extremities, and from effusion into both pleuræ; his muscular system was almost in the same state that it was in when he left the hospital in 1883. His urine contained albumen and coarsely granular casts. He improved under treatment, and left the hospital in March; but the œdema returned, and he was readmitted at the end of the same month. Ascites developed, and he died on the 14th of August, 1884.

A post-mortem examination was obtained, and the spinal cord, together with some portions of the musculo-spiral nerves, were removed, but, unfortunately, none of the atrophied muscle was kept. I examined the cord carefully, by means of sections taken from the various regions, and stained some with carmine alone, others with osmic acid and carmine.

There was no visible abnormal change in any part of the nervous matter. The nerve-tubes in the white columns were intact; there was no swelling of the axis-cylinder, or degeneration of the myelin.

The nerve-cells in the grey columns did not appear to be reduced in number, or to be at all atrophied. Their processes were well marked, and their nuclei distinct.

There was no marked increase of the neuroglia.

There was well-marked hyaline degeneration of the walls of the arteries, both in the grey and white substance of the cord. The degeneration affected, principally, the middle and outer coats of the vessels. The cellular elements were swollen, their outlines in many places being lost; the nuclei were not visible, and they did not stain with either carmine or logwood.

The musculo-spiral nerve was examined by means of transverse and longitudinal sections, and preparations were also made by teasing out the fibres with needles. The sections and teased-out specimens were stained with carmine and osmic acid.

The axis-cylinders were everywhere distinct; there was no fragmentation of the myelin sheaths, except in a few instances, where the breaking up was evidently due to the method of preparation. There was no proliferation of the nuclei of the sheaths, nor was there any increase of the surrounding connective tissue. The walls of the small arteries in the nerve-sheaths had undergone a hyaline change similar to that noticed in the arteries of the cord. The radial nerve was not kept.

The kidneys were small, the capsule was adherent, and when separated carried with it portions of the kidney substance.

Microscopically they presented, in a well-marked form, the changes of advanced cirrhosis. The newly formed connective tissue was most abundant and most dense round the interlobular arteries.

When lead-poisoning first attracted observation, attention was chiefly directed to the affection of the extensor muscles, and Gusserow[1] attempted to explain the paralysis by supposing a particular affinity of lead for the muscles; but this hypothesis was completely destroyed when Heubel[2] demonstrated the fact, that the muscles contained less lead than the bones, kidneys, liver, brain or spinal cord; and some researches undertaken by Bernhardt,[3] with a view of discovering the relative amounts of lead contained in the paralysed extensor and the normal supinator muscles, gave negative results.

No one would undertake such investigations now, for it is well known that the action of lead, as a poison, is not local but general; the general effect being evidenced by the various encephalic symptoms due to lead, by optic neuritis, amaurosis, hemianæsthesia, and other anæsthesiæ of irregular distribution; tremors, and paralysis, not limited to one set of muscles, but affecting the muscles of the body generally;[4] colic, cramps, and affections of the kidneys.

Though the paralysis produced by lead is by no means limited to the extensor muscles, still they are generally the first to show definite changes. The researches of Onimus,[5] carried out on the muscles and nerves after death, point to an explanation of this fact. He found that the extensor muscles and nerves lost their excitability before the flexor muscles and nerves, and those of the upper extremity before those of the lower. Again, the fact, first pointed out by Dr. Semon, is now well recognised, that the abductor muscles of the larynx always become paralysed before the adductors, in central or peripheral disease of its nerves. These observations point to the conclusion, that the extensor and abductor muscles and nerves possess a less power of vital resistance than the flexor muscles and nerves, and consequently, under the action of a general poison, they will be the first to succumb.

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[1] Virchow's 'Archiv,' 1881.

[2] 'Path. u. Symptom. d. Chem. Bleilähmungen,' 1871.

[3] 'Archiv für Psychiatrie,' iv., p. 616.

[4] Buzzard, 'BRAIN,' No. 1. Duplaix, 'Archiv. Gén. de Med.'

[5] "On the Excitability of the Nerves and Muscles after Death" ('Journal de l'Anatomie,' No. 6, 1880).

Concerning the lesion which is the direct cause of the paralysis three views are at present held.

Friedländer[1] holds that the primary change is a myositis, induced by the lead, and that this is followed by a neuritis, which ascends to the cord.

The second hypothesis, supported by Zunker, Leyden and Ferrier, is that the primary change is a peripheral neuritis. Lastly, Remak, Vulpian and Déjérine believe that lead produces a poliomyelitis anterior.

So far the results of the published cases appear to favour the second view. We know that lead acts injuriously on the nerve-cells in the brain, producing in many cases serious symptoms, and, not rarely, death. There is no reason, therefore, why it should not have a direct action on the nerve-cells of the cord. Further, a poison may seriously interfere with the function of a nerve-cell, and yet, at first, produce no visible change in it; alcohol and strychnia act in this manner.[2] But, while the nerve-centres are subjected to the direct influence of the poison, and must also be injuriously affected by the general arterial degeneration, the nerve-fibres are at a still greater disadvantage, for, besides being directly acted upon by the lead, and suffering from the arterial degeneration, they will also be affected by the loss of the trophic influence which, in a state of health, the nerve-cells exert over them.

Thus it appears probable, that as the nerves suffer in a double manner, as compared with their centres in the cord, alterations will proceed more rapidly in them than in their centres, and consequently visible changes will, as a general rule, be first recognised either in the nerve-trunks, or their peripheral terminations. So far the published cases support this view, for in 11 out of the 21 cases, changes were found only in the nerves and muscles.[1] In the case I have reported above, no changes were discovered either in the cord or the trunk of the musculo-spiral, though it is very probable that some degeneration would have been found in the radial nerve or its peripheral terminations, had they been kept.

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[1] Virchow's 'Archiv,' 1878.

[2] Cf. paper by De Watteville, "The pathogeny of lead paralysis," 'Lancet,' 1880.

In 4 cases, degeneration was found both in the nerves and the anterior nerve-roots, the cord not being affected;[2] while in 5 cases the cord, as well as the nerves and their roots, was affected:[3] and in one of these, namely that described by Zunker, the changes in the cord, which consisted of localised spots of softening, did not correspond to the centres, from which the diseased nerves sprang. It is to be noted, further, that, in all the cases in which visible alterations were discovered in the cord, the symptoms had been present for a long period of time.

Finally, I have to thank Dr. Dreschfeld for his kind assistance in the examination of the microscopical specimens.

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[1] Lancereaux, 2; Gombault, 1; Westphal, 1; Tiburtius, 1; Ollivier, 1; Eisenlohr, 1; Déjérine, 3; Moritz, 1.

[2] Friedländer, 1; Déjérine, 2; Duplaix, 1,

[3] Lancereaux, 1; Zunker, 1; Vulpian, 1; Monakow 1; Oeller, 1.

Clinical Cases.

CASE IN WHICH ATTACKS OF INTERMITTENT TONIC MUSCULAR SPASMS, IMMEDIATELY FOLLOWED BY COMPLETE TEMPORARY PARALYSIS, HAVE FREQUENTLY AND PERIODICALLY OCCURRED DURING THE ENTIRE LIFE OF THE PATIENT, THE HEALTH IN THE INTERVALS BEING NORMAL.

BY A. HUGHES BENNETT, M.D.,

Physician to the Hospital for Epilepsy and Paralysis, and Assistant Physician to the Westminster Hospital.

Antecedent History.—The patient is a young woman aged 17. Her family history is unimportant.. The mother states that during utero-gestation she herself suffered from nervous symptoms as the result of fright, from being chased by a mad dog. She nursed the child for a year, but it was always very delicate and undersized, and was unable to walk till it was three and a half years old.

At birth the patient is stated to have been apparently healthy, but three days afterwards she was seized with a "fit." After this first attack, which was of a convulsive nature, she had no definite illness, but was subject to frequent "faintings," in which she lay for a short time motionless, and "turned up the whites of her eyes." When three years old, it was for the first time observed that in these seizures the limbs were "drawn up." These periodical attacks have continued since, but have become more frequent and severe. Two or three years ago they were accompanied by screaming and other apparently violent hysterical symptoms, but of late these have disappeared. The catamenia have never occurred. In the interval between the attacks the physical health has been good, but mentally the patient is considered deficient, and although she has been to school, she cannot read or write like other girls of her age.

Present condition—A. *During the intervals of the attacks.*—The patient is pale and delicate-looking, and seems several years younger than her age, but all the organs and functions are normal, the body is well nourished, and the general health is good.

She has a peculiar sardonic expression, owing to the lower lip being tightly contracted round the teeth, over which the upper lip projects. There is marked flattening of the skull in the region of the posterior fontanelle. The movements of the eyeballs are somewhat restless and unsteady, and there is difficulty in fixing them on any object, but there is no strabismus or otherwise deficient action. The pupils are equal and normal, and contract to light and accommodation. Vision is natural. The mouth and face are symmetrical, but their movements are accompanied by slight choreic-like twitchings. The tongue is straight, and its actions are apparently normal, with the exception of a slight restlessness on movement. The articulation is not very distinct, but without marked peculiarity. The patient is cheerful, and willingly assists in the work of the ward; she answers questions of an ordinary kind, and conducts herself in a way which has not caused any special comment from the nurses. Her intelligence is, however, evidently defective. She is slow and stupid, childish in her manner, cannot give an account of her illness, has a bad memory, and cannot read or write. The special senses are all intact. The appearance, movements, and strength of the limbs are normal in every respect, except that there is a constant slight restless or choreic-like movement of all the extremities. The grasp of the hands and all the actions of the upper limbs are as in health. Locomotion is normal, and the patient can walk and move about like other persons. There is no rigidity or wasting of the muscles, which have a natural appearance. Sensibility of the skin is everywhere normal. The knee-jerk phenomena are distinctly increased, but the plantar reflexes are normal. The mechanical irritability of the muscles is not markedly augmented. The electrical reactions are somewhat difficult to determine with delicacy, owing to the nervousness of the patient, and to the choreic-like movements of the limbs. They appear to be everywhere normal, with the following modifications. 1st. Stimulating the *muscles* with the galvanic current causes more vigorous contraction with the same strength of current, than when the trunk of the *nerve* which supplies them is acted upon. 2nd. In both muscles and nerves the AOC is slightly but distinctly greater than the ACC; the KCC being greater than either, and KOC is not obtainable by the strongest currents which could be tolerated.

B. *Condition during the attacks.*—This case having been under observation for upwards of a year, the condition during the so-called attacks may be summarised as follows. The patient, while in her usual healthy state, suddenly, and without apparent cause, experiences a peculiar sensation which she cannot describe, but which indicates that a seizure is imminent. She at once goes to her mother, who observes "the veins in her forehead swelling." A few minutes afterwards a sudden tonic spasm of certain muscles takes place, during which they become tense and hard, and which contract the limbs in different directions, so as to cause a variety of distortions. The parts thus affected are immovable and rigid, and their position cannot be changed by voluntary effort or even by considerable active force. When the legs are thus attacked, the patient falls down, owing to the violently produced deformity which ensues. There is no loss of consciousness or intellectual disturbance, and only when the contractions are unusually severe is there pain of a cramp-like character. This condition having lasted for about five minutes, the rigidity rapidly relaxes, and is succeeded by complete paralysis in the same distribution, in which the limbs hang flaccid, useless, and incapable of any voluntary movement. This state continues for about five minutes, after which gradually the muscles regain their power, and in from ten minutes to a quarter of an hour the patient is in a perfectly normal condition. This constitutes one attack. For the past few years the rule has been for these seizures to occur at irregular intervals, many times a day for about a week, after which there is a fortnight of complete freedom, during which time the patient is quite well. Then occurs another week of attacks, and so on, with fairly regular occurrence. These seizures are not all of equal severity. Most commonly one or more limbs only are affected; but occasionally the entire body is in a state of rigidity, every voluntary muscle apparently being in a state of violent contraction. Frequently a single limb only is involved; usually both extremities of the same side; sometimes one arm and the other leg; more rarely the two arms, or the two legs; and occasionally the entire body. When the limbs are the subject of this spasm the effects are not always the same, the muscles do not seem to be always attacked with the same proportionate severity, and consequently the deformity varies, and this may occur during different attacks without apparent definite order. Some of the chief attitudes assumed are portrayed in the annexed drawings, which, however, do not give a complete idea of the intense muscular contraction which deforms the limbs. In the upper extremity there are three prominent types. 1. The upper arm adducted and rotated inwards and backwards; the forearm extended and pronated, the wrist flexed, the proximal phalanges of the fingers flexed and their two terminal phalanges extended; the thumb extended and abducted; the entire limb projecting in a straight line posteriorly, the palm of the hand looking backwards and upwards (Fig. 1). 2. The upper arm abducted and at right angles to the trunk; the forearm flexed; the wrist, fingers and thumb flexed; the clenched fist resting on the shoulder (Fig. 2). 3. The upper arm adducted, with the elbow close to the side; the forearm flexed; the wrist extended; the proximal phalanges of the fingers and thumb extended; their distal phalanges flexed; the hand resting on the shoulder, the palm upwards (Fig. 3).

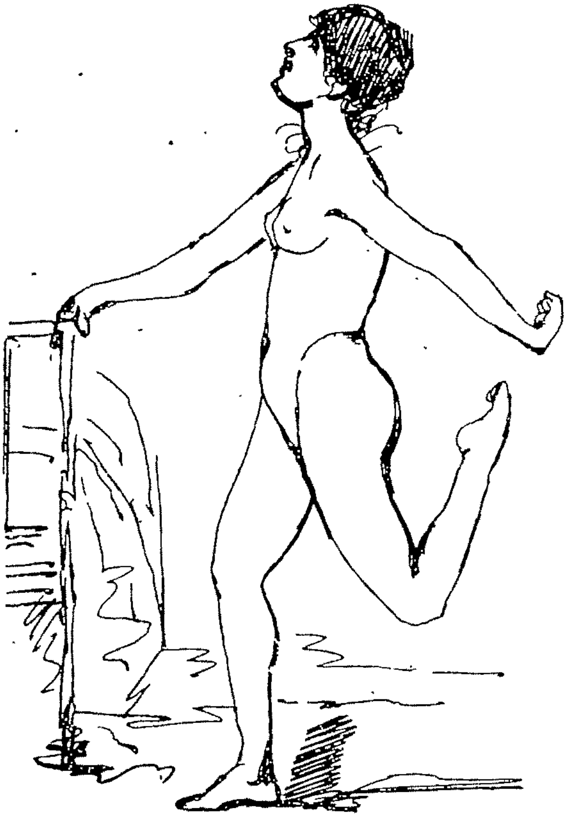

FIG. 1.

In the lower limb there are also three chief types of deformity. 1. The thigh completely extended; the leg flexed; the foot extended; the toes flexed; the plantar aspect of the foot looking upwards and forwards. (Fig. 1.) 2. The thigh partly flexed and adducted, so that the knee crosses the other limb; the leg slightly flexed; the foot inverted; the toes flexed. (Fig. 2.) 3. The thigh, leg, foot and toes extended, so that the limb is in a state of rigid straight extension, the toes pointing downwards, the foot being in the position of talipes equinus. (Fig. 3.)

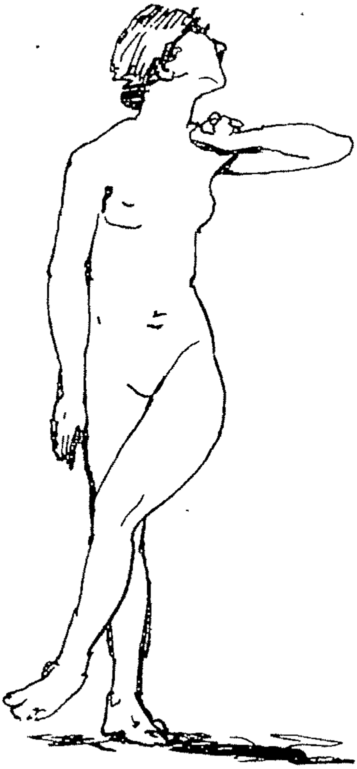

Fig. 2.

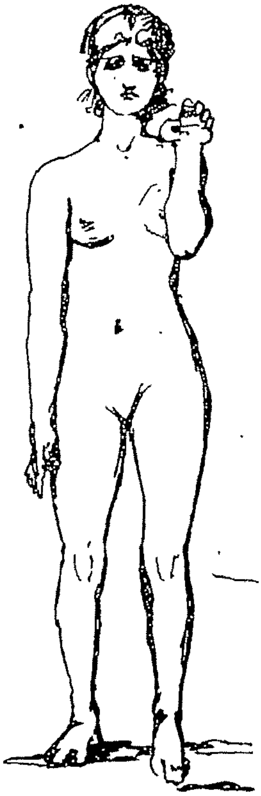

Fig. 3.

Even these three main types of deformity in the limbs undergo various minor modifications. When the entire body is attacked, one or more of the positions described of the limbs may be assumed, and are generally different in the two sides, so that a most irregular appearance is the result. (Fig. 4.) In addition, the muscles of the face are contracted and rigid, and the countenance distorted in a variety of ways; the eyelids are sometimes forcibly closed, sometimes widely open. The eyeballs generally are fixed upwards and inwards, so that only the white of the eyes can be seen. The neck is stiff and rigid, the head being generally forced backwards till it touches the back. There is evidently obstruction to the breathing, possibly due, partly to contraction of the muscles of the neck, and partly to fixation of those of the thorax. The spine is arched and in a condition of complete opisthotonus. During such an attack the senses are acute; the muscles are all hard and tense, there is severe cramping pain, and considerable dyspnœa. Swallowing is impossible, speech is prevented, and the skin is bathed in profuse perspiration. This is succeeded by complete temporary general paralysis, in which the limbs hang limp and perfectly useless. The nature of any seizure cannot be predicted, and the extent and distribution of the deformities succeed one another without method or regularity. The precise order in which the individual muscles were attacked could not be determined with any degree of exactitude on a sufficiently extensive scale to form any generalisations, as the physician himself did not frequently observe the entire paroxysm, and the statements of the nurse were not sufficiently accurate. As far, however, as could be ascertained, the most common sequence of events was as follows: Contraction begins in the left hand, spreads upwards to the forearm and arm; then involves the left foot, and subsequently the rest of the lower limb; afterwards the face, eyeballs and head, which last is drawn backwards and towards the left. No exciting cause for the paroxysms could be determined, except that they were liable to be induced by mental emotion and excitement. They never took place during sleep, and were not brought on by voluntary motion or artificial stimulation.

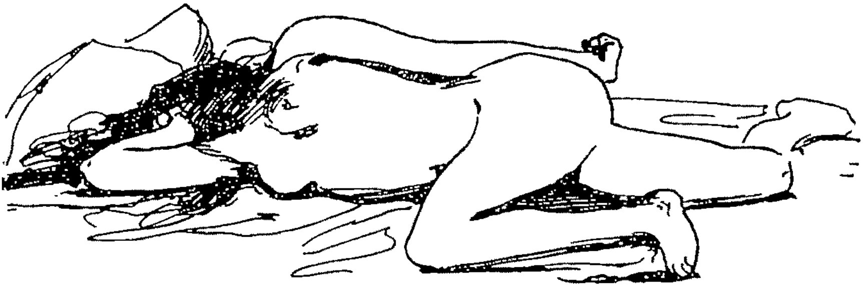

FIG. 4.

Progress of the Case.—No essential change has taken place in the last fifteen months, during which the patient has been under observation. The attacks still occur with their former frequency, but of late their severity has somewhat diminished. Treatment of a variety of kinds, including tonics, sedatives, and electricity, has been followed by absolutely negative results. During the spasmodic attack, friction of the affected parts usually gave relief. For the past few months the general health of the patient has been unsatisfactory, and there is a suspicion of incipient phthisis: otherwise her condition is as before.

Commentary.—The clinical picture just portrayed does not exactly correspond with any of those diseases to which a distinguishing name has been given. I have therefore been compelled to describe the case under the appellation of its chief and characteristic symptoms, namely, attacks of intermittent tonic muscular spasm, immediately followed by complete temporary paralysis beginning three days after birth, occurring frequently and with almost regular periodicity throughout the entire life of the patient, the health between the seizures being normal. Although the sequence of events detailed does not in all respects answer to any of the typical descriptions of our text books, it at the same time presents certain features in common with some of them. These it may be well to notice, with the view of determining whether the affection under consideration may not be a modified form of some well-known disorder.

In so-called "Thomsen's disease," for example, there are temporary attacks of cramp which may have existed during the whole life of the patient; but between this and the condition of our patient there is an important distinction to be made. In Thomsen's disease the muscular spasm is directly caused by voluntary efforts, and by them alone. In the case before us the contractions are spontaneous, and in no way affected by active movement.

To the severe forms of Tetany this case bears some resemblance. The intermittent nature of the spasm is common to both. On the other hand, these two affections diverge in many important particulars. The disease under consideration having begun immediately after birth, and continued ever since; the attacks being unaffected by external irritations and the characteristic hyper-excitability of the nerves on electric stimulation being absent; the temporary but complete paralysis after each seizure, and the impaired mental faculties; all warrant us in removing it from the typical category of Tetany.

To the contractures of hystero-epilepsy this case might at first sight appear to bear a likeness, and the annexed drawings of some of the deformities presented by the patient are somewhat similar to those delineated in the beautifully artistic representations of M. Richer in his great work.[1] Here, however, consciousness was never lost during the attacks, and there was a total absence of that train of other symptoms characteristic of hysteria. It is true that there was a somewhat vague account of the girl's conduct, lasting for a few months only, which suggested that disorder, but this, if present, was probably a complication of her already existing malady.

This case must therefore stand by itself, as it cannot be placed under the head of any of those affections in which there is a typical clinical history, condition, or progress. Although its leading features have already been detailed, I may now summarise the salient points, those which are unusual, and which render it specially worthy of attention. 1. The disease began at the early age of three days, and has continued without intermission for seventeen years. 2. The most prominent symptoms were attacks of intermittent tonic muscular contractions of varying severity, and involving varying groups of muscles. 3. The seizures of muscular contractions were periodic, and occurred at almost perfectly regular intervals during every third week of the patient's life. 4. The muscular contractions occurred spontaneously without apparent cause, and were not induced by voluntary motion, by friction or other external stimuli, and the electrical excitability of the nerves and muscles was not increased. 5. The muscular contractions were, as a rule, painless. 6. The contractions followed no regular order, and were accompanied by no constant or special deformity, such as the so-called gynecological hand. Most frequently the spasm began in the left fingers, extended from them up the arm to the face and head; from thence to the foot, and up the leg and thigh of the same side. 7. The attacks of spasm were immediately followed by complete but temporary paralysis, which was exactly confined to the same regions, and in which there was no trace of muscular rigidity or voluntary movement. 8. The general health of the patient in the intervals of the attacks was robust throughout, except the slight choreic movements on voluntary motion, and some degree of mental deficiency.

The pathology of this condition must remain for the present a matter of speculation. It is improbable that the muscles themselves are diseased. There is no evidence that the nerves are affected. The seat of lesion must therefore be situated in the nervous centres, probably in the brain, and possibly in the motor areas of its cortex. Its nature we cannot demonstrate. It is scarcely likely that any gross organic degeneration is present. We must therefore assume the disturbance to be of dynamic origin and due to delicate molecular trophic changes, the existence of which is hypothetical. Hence this disorder may be looked upon as a neurosis. The intermissions, the periodicity, and the paralysis following the spasms, would seem to indicate a continued irritation of ganglionic cells in the grey matter, which causes explosions at intervals, as evidenced by the motor paroxysms, which excess of energy is followed by exhaustion in the shape of paresis. The occasional order in which these phenomena take place is suggestive of this irritation being situated in the cortical substance of the cerebrum.

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[1] 'Etudes Cliniques sur l'Hystéro-Épilepsie ou Grande Hystérie.' Paris, 1881.

A CASE OF MULTIPLE TUBERCULAR TUMOURS, ONE OF WHICH WAS SITUATED IN THE LEFT CRUS CEREBRI AND CAUSED PARALYSIS OF THE THIRD NERVE OF THAT SIDE, WHILE ANOTHER WAS SITUATED IN THE SPINAL MEMBRANES ON THE LEFT SIDE, ON A LEVEL WITH THE JUNCTION OF THE CERVICAL DORSAL REGIONS, AND PRODUCED PARALYSIS OF THE MOTOR AND SENSORY BRANCHES DERIVED FROM THE SEVENTH AND EIGHTH CERVICAL AND FIRST DORSAL NERVE-ROOTS.

BY JAMES ROSS, M.D., LL.D., F.R.C.P.,

Senior Assistant-Physician to the Manchester Royal Infirmary.

For the notes of the following case I am indebted to my clinical clerk, Mr. O. Kauffman.

C. P., a widow aged 24 years, was admitted on December 29th, 1883, into the Manchester Royal Infirmary, under the care of Dr. Ross.

The patient states, that when an infant of about two years of age her right arm was injured by a fall. From this accident dated a protracted illness, from which she did not recover until pieces of bone were removed by operation from her right forearm. About the same time abscesses formed in the neighbourhood of the right knee, and these had to be opened. From that time forwards she enjoyed pretty good health, until four or five years ago, when her left ankle became diseased, and three years ago Mr. Lund had to amputate the leg below the knee. Soon after recovering from the operation the patient married, and had a child eleven months ago, which is now living and healthy.

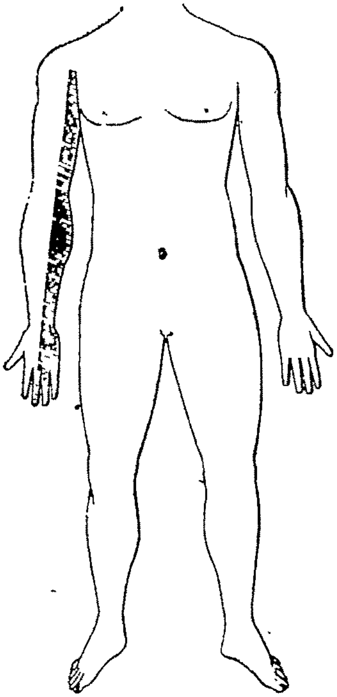
A few weeks after the birth of her child the patient began to suffer from a constant, deep-seated, throbbing occipital headache. During the following six months headache was the only symptom of which the patient complained, but at the end of that time she found on awaking in the morning that the left eyelid had "fallen down" and that she was unable to open that eye.

Soon afterwards her left hand became suddenly paralysed, whilst at the same time she felt a numbness of the inner aspect of the forearm, and the inner half of the hand, and of the little and ring-fingers.

Present condition.—The patient is a small and pale woman, with a pimply and blotched face. The back of the right elbow-joint and of the forearm is covered with large cicatrices, and the joint is partially ankylosed. The right knee-joint is also covered with cicatrices and ankylosed, and the patient is at present suffering from strumous disease of the great toe of the right foot. The left leg presents a healthy stump after amputation about the middle of the calf.

There is complete ptosis of the left eyelid, and on raising the lid by the finger, the eye is seen to be directed outwards, while the power of directing it inwards, upwards, and downwards, is almost completely lost. The pupil is in a moderate condition of dilatation. There is no ptosis of the right eyelid, but the movements of the eyeball appear to be somewhat restricted, but it is difficult to know whether this is due to an actual paralysis or to the peevish disposition of the patient, who can hardly be induced to do anything she is told. The grasp of the right hand is comparatively good, and there is no evidence that it is the subject of any paralysis. The muscles of the right lower extremity are wasted, and all the movements are feeble, but there is no evidence of special paralysis. The condition of the tendon-reactions could not be ascertained on the right side of the body, owing to the cicatrices over the elbow and knee and the ankylosis of those joints. The grasp of the left hand, the side on which the ptosis exists, is completely lost. When the patient is asked to grasp any object, the hand is feebly hyper-extended at the wrist, but the fingers do not become in the slightest degree flexed. The muscles of the thenar and hypothenar eminences, the interossei and lumbricales, the long flexors of the fingers and the flexors of the wrist are much atrophied; and even the extensors of the fingers, thumb, and wrist are wasted; so that of the muscles of the forearm and hand the supinator longus alone has retained its plumpness and power, and this is the only one which contracts when its tendon is struck. The hand presents the well-known claw form produced by atrophy and paralysis of all its muscles. The muscles of the right thigh are considerably wasted, the patellar tendon reaction cannot be elicited, and the movements of the limb are feebly performed, but it is doubtful whether this is due to general weakness or to a special paralysis. An objective examination shows that there is almost complete anæsthesia of the skin over the back and front of the inner half of the hand and two and a half fingers, and of the inner aspect of the forearm, while there is partial anæsthesia of the inner aspect of the upper arm. (Fig. 1.) There is no evidence of anæsthesia of any other part of the body, but the patient complains of pains in the lower extremities. The fundus of each eye is found to be normal on ophthalmoscopic examination, and there is no disorder of any of the special senses. The urine is acid, specific gravity 1017, and free from albumen. There is no evidence of disease of any of the internal organs.

FIG. 1.



THE SHADED PORTION SHOWS THE DISTRIBUTION OF ANÆSTHESIA IN THE CASE OF C. P., EXCEPT THAT IT IS INADVERTENTLY REPRESENTED ON THE RIGHT, INSTEAD OF THE LEFT SIDE.

Jan. 8th.—A few days after admission the urine began to dribble, and it is now passed in bed; but the fæces are still retained. The patient complains of some difficulty in swallowing, and states that she has to take a drink after every morsel of solid food swallowed. Food collects in both cheeks, so that she is sometimes obliged to remove it from between the cheeks and the teeth with the aid of the finger. Slight difficulty is experienced in the articulation of words. The nerves and muscles of the left arm react to a faradic current of moderate strength; but the extensors and flexors of the forearm, and the intrinsic muscles of the hand, contract to a weaker galvanic current on anodal than on cathodal closure, these muscles thus manifesting Erb's partial reaction of degeneration.

Feb. 9th, 1884.—Since the date of last report the patient has suffered from headache and vomiting. She is restless and wakeful at night, and shouts out so continually that she has become a nuisance in the ward. During the day she is drowsy, and can hardly be got to reply to any question. The few words which she utters are so indistinct as to be hardly intelligible. The patient was now discharged.

March 8th.—C. P. was re-admitted to-day. She is now cold and almost pulseless, and passes her urine and fæces in bed. She only mutters a few words, and these are so indistinctly articulated as to be almost unintelligible. The muscles of the right hand are now slightly atrophied, but it is impossible to subject the patient to an electrical examination. She gradually sank, and died on the night of her second admission.

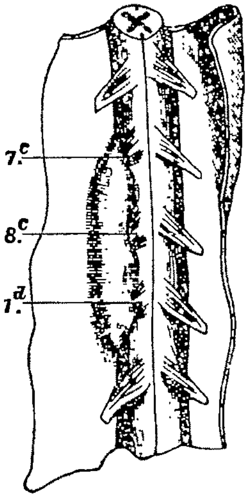
Autopsy.—The post-mortem examination was conducted by Dr. Maguire on March 10, about 36 hours after death, and the following is his report: "The body is much emaciated. Rigor mortis present—post-mortem-staining on back. Pupils equal and of ordinary size. Orifices of body normal. Left hand is claw-form, and shows atrophy up the thenar and hypothenar eminences. Right arm shows old cicatrices about elbow and ankylosis of the joint. The hand is also claw-shaped, and there is atrophy of the thenar and hypothenar eminences, but less marked than on the left hand. Left leg amputated below the knee, and wound completely healed. Right knee ankylosed, and shows old cicatrices about the joint. Subcutaneous fat scanty. Both pleuræ show old adhesions. The left lung shows some patches of pneumonia in stage of hepatisation near the root. The right lung is congested, but otherwise normal. No tubercles to be seen in any part of the lungs. Pericardium normal. The right heart contains post-mortem clots, the left heart is empty. Valves normal.

"The abdominal organs did not show any noteworthy changes. The brain is somewhat congested, and on separating the anterior and middle lobes of each side, small miliary tubercles are seen in each Sylvian fissure. Near the origin of the left Sylvian artery the vessel is surrounded by a hard tuberculous mass, which almost obstructs it. On section, a tuberculous nodule of the size of a large pea is found in the left crus cerebri. It lies in the crusta at about the middle of its length.

The nodule is generally hard, but shows a few spots of caseous degeneration. A similar tuberculous nodule is found in the right crus cerebri, but is situated in the tegementum, and on a much higher level than the nodule in the left crus. The ventricular fluid is increased, and the choroid plexus injected.

"Several tuberculous tumours are found in the spinal cord. One is situated on the right side between the second and third cervical nerves; another on the left side between the seventh cervical and first dorsal nerves (Fig. 2); while a third is situated over the centre of the lumbar enlargement. The tumours seem to the naked eye to grow from the membranes, and to pass inwards in the cord. Patches of thickened membrane are found in many parts of the cord."

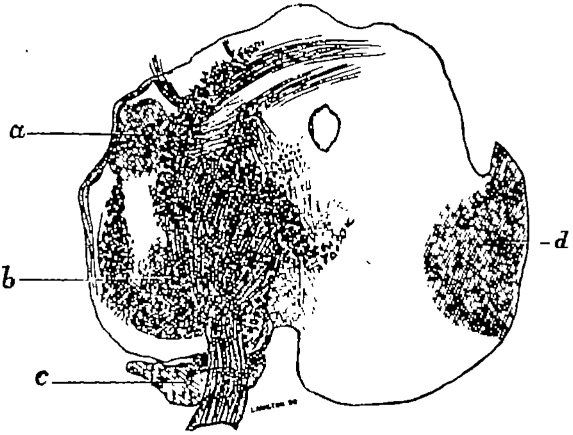
Fig. 2.



Portion of Spinal Cord, showing Tumour in Lower Cervical and the Upper Dorsal Region.

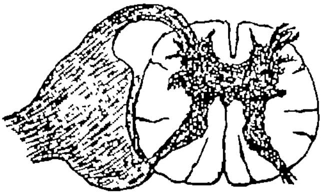
The microscopical examination was kindly conducted by my friend Dr. Larmuth, and the annexed diagrams are taken from sections of the crura cerebri (Fig. 3) and of the spinal cord on a level with the eighth nerve (Fig. 4). In the left crus (Fig. 3), a nodule of tubercle (*a*) is seen in the tegementum in such a position that it does not interfere with the third or fourth nerves; a second somewhat larger nodule (*b*), which involves the fibres of the third nerve and the motor tract, is situated in the crusta; while a third nodule (*c*) implicates the root of the third nerve after its emergence from the crus. This nodule was distinctly felt at the autopsy, although it is not mentioned in Dr. Maguire's report. In the right crus a large nodule (*d*) is situated in the tegementum, but it does not involve either the third or fourth nerves or the motor tract.

FIG. 3.



(LARMUTH.) FROM A SECTION OF THE CRUS CEREBRI, SHOWING THE POSITIONS OF THE TUMOURS.

FIG. 4.



(LARMUTH.) FROM A SECTION OF THE SPINAL CORD ON A LEVEL WITH THE RIGHT CERVICAL NERVE, SHOWING THE RELATION OF THE TUMOUR TO THE NERVE-ROOTS.

In the section of the spinal cord the tumour is seen to be attached to the membranes over the posterior part of the lateral column, and to involve both the posterior and anterior roots of the nerve. The tumours consisted of lymphoid cells imbedded in a fibrillary matrix, but it was not thought necessary to search for bacilli, as the interest of the case centered in the localisation of the lesions.

Remarks.—My diagnosis of this case from the first was that a tubercular tumour was situated about the root of the third left nerve at its point of emergence from the crus; that a second involved the anterior and posterior roots of the seventh and eighth cervical and first dorsal nerves, and that tubercles were scattered over the lumbar region of the cord, some at least of these being situated at the upper part of the lumbar region.

The slow and progressive manner in which the symptoms supervened, rendered it more likely that the lesion would be some kind of growth, and not a rupture or occlusion of a blood-vessel; while the presence of cicatrices, ankylosis of joints, scrofulous disease of the great toe of the right foot, and the evidences of an amputated leg, showed unmistakably that the growth would be tuberculous.

With regard to the localisation of the lesion, the fact that disease of the left third nerve was unaccompanied by disorder of the functions of any of the other nerves which pass through the cavernous sinus, or by hemianopsia from implication of the optic tract, showed that the tumour was not compressing the nerve in its course along the base of the skull; the absence of any evidence of hemiplegia of the right side of the body led me to believe that the nodule would not be situated in the crusta; therefore I concluded that it would be found at the root of the nerve after its emergence from the crus. It was thought at one time that there were slight evidences of paralysis of the third nerve of the right side, and this induced me to believe that we should find the tubercular deposit extending across the interpeduncular space to reach the root of the nerve on the right side. The evidences of paralysis of the right third nerve were, however, always doubtful, as it was probable that the restricted movements of the eyeball were due to the mental condition of the patient.

My anticipations with regard to the localisation of the tumour which caused the paralysis of the third nerve were not in all respects confirmed at the autopsy. It is true that a nodule was found at the point of emergence of the left third nerve, but I was very much surprised to find that another nodule was situated in the crusta, just in that position in which it might be expected to cause a spasmodic paralysis of the extremities on the opposite side of the body by interference with the pyramidal tract. Several reasons may be given for the absence of any evidence of right-sided hemiplegia of cerebral origin. There was no opportunity for examining the patient carefully after her discharge from the hospital on Feb. 9th, a month before her death, and it is therefore possible that a right-sided hemiplegia may have supervened in the intervening period. And even a certain degree of right-sided hemiplegia may have existed before the date of the patient's discharge from the hospital without having been detected, because the phenomena of a spasmodic paralysis would be greatly masked in this case by the presence of other symptoms. The reactions of the tendons of the triceps and of the patella were abolished on the right side, owing to local disease of the elbow and knee-joints; the periosteal reactions of the radius and ulna would be lost partly from old-standing disease of the bones, and partly from a certain diffused atrophy of the muscles which even existed on the right side; and ankle-clonus would also be arrested from the diffused atrophy of the muscles of the lower extremity which was present; while a feeble grasp, and feeble movements of the limbs generally, might only be recognized as an expression of general weakness. But it is possible that right-sided hemiplegia had not existed at any time, for it is well known that a tumour of considerable size may encroach upon the pyramidal tract without causing paralysis, the fibres in such cases being pushed aside instead of being compressed or completely ruptured.

On observing for the first time that the third nerve and the hand were paralysed on the same side of the body, the first idea which suggested itself was that the case was an example of a direct instead of a crossed cerebral paralysis; but even a cursory examination sufficed to dispel this notion. The muscles of the left forearm, with the exception of the supinator longus, failed to react when their tendons were struck at the wrist, and these muscles with the same exception, as well as the intrinsic muscles of the hand, were found atrophied, and manifested the "partial reaction of degeneration" on electrical exploration. The hand also assumed the claw form which is met with in cervical pachymeningitis when the disease is on a level with the eighth cervical and first dorsal nerves, with the exception that hyper-extension at the wrist was not a marked feature of the case, inasmuch as the extensors, as well as the flexors of the wrist and fingers, were involved in the paralysis and atrophy. The implication of the extensors showed that the seventh cervical nerve was also involved in the disease, and it was therefore concluded that the lesion involved the motor roots of the seventh and eighth cervical and first dorsal nerves. The conclusion reached, from a consideration of the motor disorder of the left hand and forearm, was in the main confirmed by the sensory disorder observed, the anæsthesia being present in the area of distribution of the nerve of Wrisberg, the internal cutaneous, and the sensory branches of the ulnar, these nerves being derived from the eighth cervical and first dorsal nerves.

It will be remembered that the anæsthesia of the inner surface of the upper arm was much less complete than that of the corresponding surface of the forearm, which is explained by the fact that the former surface is supplied by the intercosto-humeral nerve, which is derived from the second dorsal, the root of which was not involved in the disease.

Having once concluded that a tubercular tumour was situated in or near the crus cerebri; that a separate lesion, situated in or near the spinal cord, involved the anterior roots of the seventh and eighth cervical and first dorsal nerves, and the posterior roots of at least the last two of these nerves; and that the lesion was chiefly unilateral, there was no difficulty in inferring that the nature of the spinal lesion was tubercular; that it formed a more or less localised growth in the spinal membranes; and that the roots of the implicated nerves passed through it. These conclusions were amply verified at the autopsy. The existence of tubercles in the lumbar enlargement of the spinal cord was suspected from the presence of a diffused atrophy of the muscles of the lower extremities, and, in order to account for the incontinence of urine, it was assumed that some of these were situated in the upper part of the lumbar region.

The presence of the tumour in the spinal cord on a level with the third nerve was not suspected during life; but this is not surprising, when it is considered that the patient was unable to sit up in bed, and that consequently disorders of the innervation of the cervical plexus, difficult to detect under any circumstances, would be likely to escape notice.

The disorders of speech were attributed during life to interference with the function of the geniculate fasciculus of the pyramidal tract in its passage through the crusta; but we are now inclined to regard them as resulting from anæmia of the motor area of the left hemisphere, caused by the tubercular growth which surrounded the point of origin of the left middle cerebral artery.

CASE OF INJURY TO RIGHT SIDE OF HEAD, FOLLOWED BY COMPLETE PARALYSIS OF RIGHT ARM.

BY GEORGE THOMSON, M.D.,

Surgeon, Oldham Infirmary.

J. M., aged 34 years, was among a crowd watching a fire on the morning of December 19th, 1883, when the fire-escape, in passing up the street, came in contact with an overhead telephone wire, causing it to dislodge a quantity of masonry about twenty feet above the heads of the people. A heavy coping-stone was among the debris, and seems to have fallen on the heads of several men, severely injuring three, of whom J. M. was one. I saw him immediately after the accident, and found him suffering from symptoms of concussion of the brain, with a scalp wound about 1½ inch long on the top of the head over the right supero-antero-parietal area of the skull, which was denuded of periosteum to about the size of a shilling. No other injury to the head was apparent.

The early symptoms were quick, small pulse, cold surface, vomiting, and partial unconsciousness. When put in bed, he lay on his side with his limbs flexed, as in cerebral irritation. Towards evening, the pulse became slow (58) and the temperature slightly rose (100°), and the man passed into deep stupor, with equal pupils, slightly contracted, but reacting to light. His condition remained practically unchanged for two days, when (Dec. 21) he began to swallow a little. During the following night he had five or six clonic spasms, lasting a few minutes, beginning in the right face and extending to the right arm.

Dec. 22nd.—These spasms occurred several times during the day, but during the intervals he was more conscious, swallowing liquids freely, and addressing his sister once by name. There was no apparent paralysis of arm or face.

Dec. 23rd.—During the night the clonic spasm returned, accompanied by clenching of the hand and jaw, with cold surface and general collapse. This condition lasted about three hours, and when it passed off, the right arm appeared to be completely paralysed. He became more conscious during the day, took liquids well; was aware of the presence of strangers, and put out his tongue when asked. The spasm recurred in the right face during the day, but not in the arm. The pulse was still under 60, and the temperature had fallen to normal. The leg was moved about quite freely. Dr. Ross saw him with me in the afternoon; and, notwithstanding the puzzling fact, that the paralysis and the external injury were on the same side, agreed that in case the circumstances became desperate it would be well to trephine where the skull was denuded of periosteum.

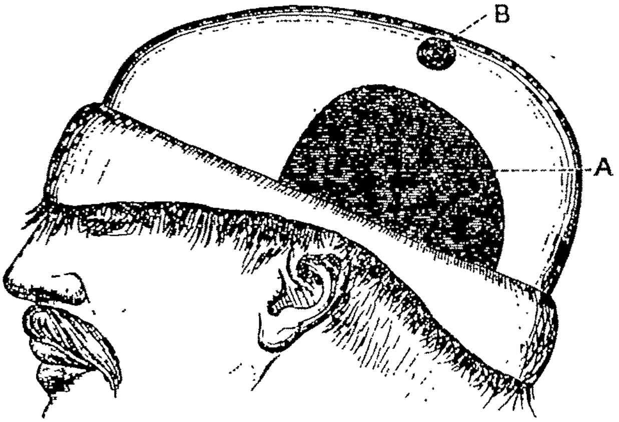


Fig 1.

Dec. 25*th.*—The pulse had gradually risen to 80, temperature 100°. He named people in the room, but had intervals of violent delirium.

Dec. 26*th.*—He was more continuously violent. Evening pulse 120, temperature 101°; pupils less contracted, and dilate widely when he is roused. Ice applied to the head.

Dec. 27*th.*—He had become so seriously worse, that I decided to try the expedient of trephining, in the hope of affording relief. I removed the circle of bone which was denuded of periosteum, and finding the dura mater very tense, incised it, liberating a considerable quantity of serous fluid. A slight relief seemed to follow, but he died during the day.

Dec. 29*th.* — *Autopsy.* — Assisted by Dr. Corns and Mr. Holton, I made an examination of the head. On reflecting the scalp, there was considerable extravasation of blood over the left parietal surface, and also in the temporal muscles. It was noticed in removing the skull-cap, as it had also been in trephining, that the bone was unusually thick and tough. An extensive crack was found in the skull, describing two sides of a triangle, with the apex about ½ inch in front of the scalp-wound. One ramus of the fissure extended forwards in a diagonal direction across the vertex, and terminated in the roof of the left orbit. The other ramus extended laterally, traversing the left supero-antero-parietal, infero-antero-parietal, and squamous areas, and terminating in the petrous portion in the floor of the middle fossa of the skull. External to the dura mater on the left side was a large venous clot (Fig. 1, A), apparently due to oozing from the diploë, and corresponding to the squamous, infero-antero-parietal, and infero-postero-parietal areas. The dura mater was lacerated for about 1¼ inch immediately under and in the direction of the fissure in the skull (Fig. 1, B), about ⅓ inch to the left of the middle line and over the ascending frontal convolution. The cortical substance of this convolution was also lacerated for about ⅓ inch immediately underneath the tear on the dura mater, and contained a small clot about the size of a pea. (Fig. 2, A.)

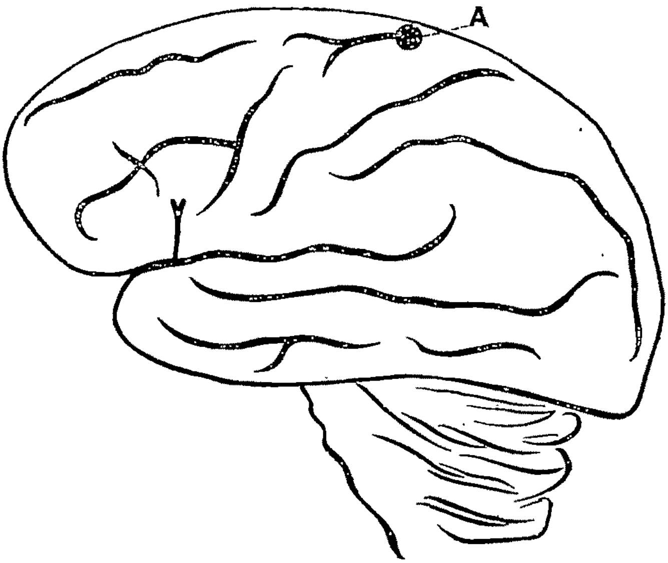


FIG. 2.

Remarks.—The most striking feature of this interesting injury, is the fact that paralysis was present on the right side, while the injury, so far as evidence was forthcoming before death, was on the same side. In point of fact the paralysis was the only reliable indicator of the portion of brain injured, the marks of external injury being quite misleading, as the post-mortem appearances show. It is certainly very unusual and extraordinary that so extensive a fissure of the skull should bear such relation to the situation of the blow, and that the point of injury to the brain and its membranes should be neither at the site of the blow nor the point of contre-coup, but at a point on the left side nearly corresponding to that on which the blow fell on the right.

Critical Digest.

ON VERTIGO.

BY GEORGE PARKER, M.A., M.D. (CAMB.)

Vertigo, or giddiness, is a symptom that almost every one has experienced under some circumstances or other, and often under such widely different conditions, that one is tempted to believe there can be no common cause. We are met at the beginning by the difficulty, that it is a purely subjective phenomenon, and therefore out of the range of many means of inquiry. We can, however, cross-examine our consciousness on the one hand, and on the other register the physical signs correlative to giddiness, such as certain forms of inco-ordination and forced motion. That they are correlative seems admitted on all hands, nor is this negatived by the fact that either may occur without the other. For ideas of action are not necessarily carried out into effect, and the mental states producing action or produced by sensation need not rise into the sphere of consciousness. Vertigo, then, may be closely related to certain movements, though it does not always accompany them.

It varies from a mere sense of confusion, which perhaps ought not to be included under the name, to (*a*) a sense of all things moving about me; or (*b*), my moving while they are fixed; or (*c*), finally a definite sense of forced motion in a given direction, which motion is actually carried out. It is not every sense of motion which produces it. If I leap, jump down a hole, dive, or fall off a chair, there is a sense of motion; but giddiness does not necessarily appear, it seems to involve a sense of abortive effort.

If then it correspond to a *negation*, a *failure* of action, or co-ordination, can we get any definite result by introspection or by judging the physical result?

We may not be able to make clear that which in itself is confusion or failure to act; but its cause may still be discovered, and it may itself be compared with the normal action, of which it is the distortion.

Vertigo is generally defined as "the consciousness of disturbed locomotor co-ordination," or, as Dr. H. Jackson says, "a rudimentary disorder" of the same; or if, with Ferrier and Goltz, we distinguish the allied functions of equilibration and locomotor co-ordination, it is the consciousness of disturbance in equilibration.

It implies a loss of proportion between the efforts to maintain the erect posture, whether real or ideal.

This generally depends on a conflict of impressions, clearly not of all kinds, for giddiness does not follow if one hand reports heat and another cold. Nor do the conflicting impressions of the insane make them necessarily giddy, nor does a contradictory logical proposition produce it. It seems that at least four senses—touch, sight, the muscular sense, and the sense of equilibrium, perhaps also that of hearing—are employed in maintaining the erect posture; and if one of these contradicts the others, inco-ordinated impulses may be sent out, from which giddiness follows. Thus a person feels giddy on looking over a precipice; for the sight of the void beneath contradicts his muscular sense, and irregular efforts follow producing giddiness.

Again, without any impression upon a sense-organ, but by acting on parts of the central organ concerned through electricity or disease, there results failure of equilibrium and giddiness. Thus vertigo is not, I think, directly the correlative of conflicting sensation, but of abortive action, and the closeness of the connection is shown by the fact, that when giddiness is very great and actual motion appears, it always is in the direction which the whirl of the giddiness took.

I shall try to show that the vertigo of ordinary clinical cases depends on distortion of normal impulses, and is caused by injuries to different parts of the same machinery, i.e. of equilibration.

The diseases in which giddiness occurs are endless, and the question, "what can be the normal functions thus interfered with in so many directions and resulting thus in giddiness?" would lead us, in the absence of other proofs, to expect a *constantly* used mechanism, such as that for maintaining equilibrium.

To commence with a frequent cause, going to the root of the matter—*lesions of the cerebellum*. There are a great number of cases on record, of injuries, growths, and degenerative changes in this organ, followed by vertigo. Though its functions are not quite clearly determined, it seems on the whole to act as a great motor centre; sometimes from unfelt impulses, as the centre of a reflex chain, and sometimes at the[1] instigation of the more complex changes which are accompanied by consciousness, and in conjunction with consciously directed movement. Finally, it has, among other functions, that of co-ordinating distant and unlike movements of the neck, trunk, and limbs, which maintain our balance.

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[1] Three difficulties may be mentioned in fixing the functions of the cerebellum. 1. Irritation may spread to other organs, such as the cerebrum. Brown-Séquard even maintained that the cerebellum is not the agent for the co-ordination of movements at all, and that we must look elsewhere; but this view of his finds few supporters. 2. The possibility of the other half taking on the whole work when one lobe is disabled. But this, though true of the cerebrum, seems opposed to the mode of action in cerebellum. 3. The possibility of the cerebrum being substituted for it when diseased or injured. What is its normal function? Bastian insists that it is the supreme organ for the reinforcement and regulative distribution of the outgoing currents, not only for co-ordination. Luys, that it engenders and distributes the force needed for movements. Flourens, Longet, and others, that it co-ordinates. Broadbent regards it as co-ordinating movements guided by vision, which subserves it on one side as the muscular sense does on the other.

To consider the *forced movements* seen in some diseases, or artificially produced—while removal of the deep layers of the cerebellum produces general loss of co-ordination without motor paralysis, section of the middle peduncle gives rise to forced rotation round the longitudinal axis, and to nystagmus. Now the first is seen in some diseases, where the patient has severe giddiness; and the second will of itself produce giddiness, as in some eye-complaints. Again, injury of the anterior part of the middle lobe is followed by rolling forwards; of the posterior, backwards; of the lateral lobe on one side only, by rolling sometimes to that side, but sometimes to the other, which may depend on interference of conscious action. So, too, disease of the vermiform process causes a rush forwards; and generally, in tumours and hæmorrhage in the cerebellum, there is staggering and giddiness of a continued non-paroxysmal type. If in disseminated sclerosis one of the patches should affect this organ, giddiness occurs, which, Charcot says, is generally gyratory; the patient and all objects seem to revolve together. This occurs in three-fourths of the cases of disseminated sclerosis, but must not be confused with that caused by the nystagmus present. If a galvanic current be sent through the mastoid fossæ from right to left, giddiness follows, the head and body sink towards the right, and objects seem to revolve from right to left. If the eyes are closed, all support beneath the left side seems gone at the closing of the current. The eyes too move towards the right, and these same events occur if the actual cerebellum be stimulated. Again, if we are turned round rapidly from right to left, objects seem to fly from left to right; as this produces giddiness, we bend our body, and turn our eyes to the right, to neutralise the sensation. This may explain the galvanic results, showing that the right side of the cerebellum co-ordinates the mechanism, which prevents displacement of equilibrium to the opposite side, and *vice versâ*.

Yet occasionally the entire cerebellum has been removed without obvious loss of equilibration, and Dr. Oliver gives a very instructive case of a tubercular tumour in the left lobe, with vomiting and periodic headaches. The young woman, however, played the piano, and *walked about* day after day, a short time before her death. He suggests that here was a substitution of the cerebrum, and conscious adjustment for automatic; that she reverted to the condition of a learner, making the co-ordinations necessary, as she had done at a time when they had not become automatic.

Weir-Mitchell found that pigeons recovered in the course of some months their co-ordinating powers after extirpation of the cerebellum, by apparently relearning the processes.[1] So too in the case of a girl recorded by Cruveilhier, where the cerebellum was atrophied. Tuke mentions several other cases of atrophy without inco-ordination. After all, we know that we can replace automatic action by conscious, when the former has broken down. Thus in crossing a plank, we can, by an effort of mind, force ourselves to stand firm; the drunkard pulls himself together with an effort, after his automatic power is gone.

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[1] The exercise of this conscious balancing caused them great fatigue and exhaustion owing probably to the constant attention required.

It may be said, that the cerebellar disease in any of these cases merely caused an irritation elsewhere; but there is no other such place discovered, the lesions causing great giddiness and inco-ordination appear often too small and chronic to affect, for instance, a distant part of the cerebrum, and in any case the cerebellar lesion is a link in the chain of causation. It may be necessary to remark here, that while co-ordination does appear to depend on the cerebellum, we have no reason to think that giddiness can occur without cerebral charges. Co-ordination may go on after the cerebral lobes are removed, and therefore, when consciousness has ceased, and giddiness has become, so far as we know, impossible.

Though irritation of the cerebellum produces giddiness, I do not for a moment suggest that it is a sign of cerebellar disease only, for it may accompany almost any brain-disease, though indeed most usually associated with cerebellar. To take the 73 cases of abscess in the brain which Sir W. Gull gives. I find, upon analysing them, only one case of vertigo which could be traced to cerebellar disease, out of 8 in which it was present. Again, Dr. Bradbury refers to a pyæmia where sudden vertigo came on, and the *post mortem* revealed plugging of the middle cerebral. Still all these cases may be looked on as indirect stimulation of the cerebellum through the associated tracts, such as the optic lobes; there is nothing in them contradictory, at least, of Ferrier's results.

How does an affection of the brain act so as to cause vertigo? The answer usually made is—By disturbance of the cerebral circulation, stimulating the nerve matter to irregular explosions. And here, after the analogy of respiratory convulsions, which are produced by deoxydised blood, acting upon one spot alone, we should expect only that definite area to be acted on where we can produce like effects by puncture or stimulation. Positive proof is wanting, because it is difficult to isolate part of the circulation;[1] but I hope to say more about this, later on. Again, how does the circulation affect the brain-substance? This would seem almost as crude as the old theory, that rotation caused vertigo by dislodging bits of brain substance, unless we have a specialised organ in which changes may be induced. Such an organ does exist and produce vertigo, both under disease, and when we experimentally alter the tension of the fluid in it. This tension, in turn, is held by many authorities to vary with the blood-pressure.

The next class of diseases is *the auditory;* and here we find much that will serve to explain the real nature of vertigo. It is a common experience that, in examining the membrana tympani, some degree of giddiness is produced, especially in syringing or if the ear be blown into. Again, the pressure of wax causes sometimes most severe vertigo; and Hughlings-Jackson describes a case when a powerful soldier was rendered almost incapable by a louse scratching on the membrane.

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[1] Dr. Bradbury's case, quoted above, comes near doing so, but the vertigo may have sprung from the pyæmia and endocarditis long before the plugging took place. Cases where vertigo follows on pressure upon the carotid may depend on interference with the cardiac depressor nerve, as in Czermak's case, or on compensatory dilation of the vertebral.

Still more serious are the occasional effects of a catarrh, or inflammation of the middle ear or Eustachian tube, and by which the stapes is pressed in upon the foramen ovale. But the most important case for us is the condition in Ménière's disease. A healthy patient is suddenly attacked by fits of tinnitus, vertigo, nausea and vomiting, but without loss of consciousness. He tends to fall towards the affected side; the attacks become more and more frequent. A patient, whose case I examined day by day, complained first of a sudden pain in the head, as if crushed under weights; next, giddiness, when the ground seemed to rise up on his right side. He could hear a watch 2 feet from the left and at not more than 2 inches from the right ear. After his paroxysmal exacerbations, as Charcot calls them, he often falls into a sweat. The usual course is that, after some years, the noises and vertigo cease, but the patient becomes stone-deaf. There is in the true Ménière's disease, an invariable coincidence of vertigo, tinnitus, deafness (especially perosseous), with more or less of nausea and syncope; and occasionally there is nystagmus, discoloration of the hands, and stiff or wry neck, but no other sign of disease.

The tinnitus is much complained of, and rises often to a shrill scream before a paroxysm. As to the ætiology, it seems pretty certain (1) that disease of the membranous semicircular canals is the cause of the symptoms; (2) that a definite brain area is affected beween which and the canals there is connection by continuity of nerve-fibres only; and (3) that these fibres are distinct, may really be considered as a 13th pair, and have nothing else to do but to form this connection. For not only is there found on dissection an inflammatory exudation, more or less solid, in the canals, and (as if to mark the identity of its mode of action with cerebellar stimulation) not only is there a tendency to fall in opposite directions, according as the lesion is irritative or destructive, but it has been pointed out that the group of symptoms, minus vertigo, do not mean disease of the canals at all, but of the middle ear and cochlea,[1] and that vertigo again depends on certain fibres of the 7th pair, which run from the canals to the superior nucleus and then to the cerebellum. Indeed, it would be contrary to analogy for such a nerve of special sense as the real auditory to produce anything but its own sensation of sound. Tinnitus, deafness, and what Dr. Jackson terms the "vital" effects, such as syncope, depend on the other or true auditory fibres running from the cochlea to the inferior nucleus in medulla. Since this nucleus is close to those of the vagus and spinal accessory, the vital effects can be accounted for as an overflow from one nerve to another, in so far as they are motor, or as a transference (like that of hip pain to the knee), if they are sensorial.

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[1] Macbride, 'Guide to the Study of Ear Disease,' p. 149. Cochlitis alone produces no giddiness, though some forms of it frequently follow affections of the middle ear.

This is confirmed by the fact that vomiting does not occur in mild cases, and is always subsequent to the vertigo in auditory disease, since the nervous discharge must be of a certain strength to overflow its track, which in any case would be first affected.

Here then is an organ for the production of vertigo as definite as that of vision. Can we account for all stomach and circulatory vertigoes by its means?

Let us examine Dr. Woakes's theory. While he accepts the ordinary explanation of vertigo in Ménière's disease, he teaches that the paroxysms are caused by irritations in the heart, stomach and arm. These are conveyed by the inferior cardiac nerve, and by branches from the brachial plexus and vagus, to the inferior cervical ganglion, which sends filaments, probably vasomotor, to the vertebral artery, and this in turn supplies the semicircular canals by one of its branches, and regulates the fluid in them. This well explains those cases in which symptoms of Ménière's disease exist without any organic ear-affection, while the accompanying fainting and nausea are primary, and the causes rather than the effects of the vertigo. Where a lesion of the canals exists, such a shock from the viscera will reinforce it, causing the paroxysmal "fits," as my patient used to call them. Of course these paroxysms could be accounted for by changing conditions of the nervous system, or by the so-called summation of stimuli. There is no special means of explaining by Woakes's theory why vomiting should precede the vertigo, when stomachal and not auditory, and should follow it when of auditory origin, and this the radiation doctrine does.

He illustrates the effect of the nervous chain by certain shot-wounds of the brachial plexus which occurred in the American war. Patients were observed on receiving them to fall suddenly to the ground without loss of consciousness, and probably from changes in the canals.

The great interest to us is that it so well explains vertigo *à stomacho læso,* and the forms which occur in heart-disease, and after many poisons where no lesion of the canals exists. He would even exalt vertigo to the rank of a beneficent function, intended to lay men in the best position to recover from faintness.

Again, he illustrates the reflex action of the ganglion by noting the congested hands seen in tobacco-poisoning; but this congestion occurs in Ménière's disease, and its occurrence discredits his argument. For though dilatation of the vertebral may be governed by the inferior cervical ganglion, it is hard to see how changes in the canals or vertebral can affect the ganglion, and through it the vessels of the arm. Dr. S. Mackenzie is my authority for the fact. I have not, I regret, yet noted a case in the aural department at St. Bartholomew's.

After all, there is no crucial experiment by which Woakes's theory can be tested. Who has stimulated the inferior cervical ganglion and produced vasomotor changes? The nearest approach to it is Dr. Chapman's water bags, where, as he asserts, he can stop bleeding from lungs or nose by hot-water bags to the cervical spine, and cause an afflux to the head by applying ice bags to the same place. I need not do more than refer to Claude Bernard's analogous experiments.

Again, how can we limit the effects to such a definite area as the labyrinth, or how do we know that an increased blood-pressure affects the fluid in the canals? I suggest that the auditory artery alone of the branches of the vertebral runs through minute bony channels in conjunction with a nerve, and that dilatation of the artery causes pressure on the nerve.

Finally, Woakes does not throw any light on the vomiting and fainting which accompany constant vertigo in the most severe form of Ménière's disease, where no heart or stomach trouble exists.

Of course it is difficult to prove such a negative as this non-existence, but it seems to me that we cannot drag in a stomach or heart irritation in every case.

Certainly if we regard the theory as merely one of several possible ways in which giddiness can be brought on, it explains Dr. Foster's difficulty, that there is often disease of the canals without vertigo. For if we are inclined to admit, that there is a centre for equilibrium like those of respiration and vomiting, and that it is to a great extent dependent on the semicircular canals, then Foster's case admits of solution in two ways. First, the disease may be so mild as to produce no vertigo without helping current from the inferior cervical ganglion, and this with healthy organs may not occur. Secondly, the other senses that contribute to equilibrium may be in some cases sufficient to replace the aid given normally by the canals. In an early case mentioned by Russell Reynolds 30 years ago, a Dr. Wollaston describes a vertigo which used to seize himself on rising from a bent posture, but *only* when he was fatigued or weakened. Whether he had really ear disease or not, the vertigo only came when the central organ was in an abnormal state.

Foster mentions, though he doubts the correctness of the observation, that vertigo can still be produced by rotating an animal after section of both auditory nerves, arguing that sensations from the auditory canals cannot be the sole cause of this kind of vertigo. This experiment, however, disagrees with an important one by Dr. James, of Harvard. He examined a number of deaf mutes to see how far vertigo could be caused in them by rapid rotation. Out of 200 healthy students of Harvard taken for the purpose of comparison, only one was free from giddiness after rotation in a swing with the eyes closed: whereas of the deaf mutes (519 in number) 186 remained perfectly free from the feeling, and 134 were only slightly affected. Unfortunately the actual lesions cannot be ascertained in all cases of deafness. Another curious fact was, that of 15 deaf mutes who had been to sea, only one had suffered from sea-sickness. To sum up: inco-ordination is rarely seen in congenital deafness; it ceases in Ménière's disease with the subsidence of inflammation, unless, as some physiologists say, the cerebellum has been involved by an ascending mischief; and I think we must confess, after Dr. James's experiment, that there is no reason to think vertigo can be caused after destruction of the auditory nerve by anything like rotation which does not affect sight or the muscular sense.

Before going to the other diseases in which giddiness occurs, we will pause to consider the normal action of the canals. I do not know how we can escape the conclusion which has been felt since the time of Flourens, that they concern the sense of equilibrium and direction in space. Passing by the stories that the savage huntsman is able to find his way by some such sense of direction, there is much evidence that when one is placed blindfold on a rotating table, it is possible to judge of the direction and angle gone through. Moreover, and this is of interest to us, if in a healthy pigeon they be cut through,

(1) It moves its head from side to side constantly.

(2) It loses the power of co-ordinating movements.

(3) It has not paralysis, uncontrollable impulses, or hallucinations of sight or hearing, as far as can be judged, but seems simply "dizzy."

(4) If a rabbit be operated on, there is also nystagmus.

(5) If only the superior vertical canal be injured, the animal falls forward; if the post-vertical, he falls backwards; and if the horizontal one, to the side of the injured canal.

There is a striking parallel to the results obtained from the cerebellum, and shows the closeness of their connection, whether we believe the canals originate for us the idea of space in three dimensions or simply register movements. Indeed I am hardly now concerned with the question apart from the vertigo of rotation. It is said that if the canals be drained very slowly, no vertigo is produced, and vertigo in Ménière's disease ceases with the active inflammation of the canals. Thus we may conceive of them as serving no purpose normally whatever towards equilibration, and yet, from their intimate connection with its especial organ, as capable of collecting from various parts and transmitting to it shocks sufficient to disturb it profoundly. Just as the wire of a battery placed in my mastoid fossa conducts disturbing currents, though not a normal part of my organism, so sudden section or quick rotation may still stimulate the canals to the production of such disturbances.

We must now pass to other clinical forms; but as we have been compelled to mention *Vertigo à stomacho læso*, as Trousseau calls it, we may consider it for a few moments. Some warm morning it happens that a man engages in business for 2 or 3 hours after a hearty and rich breakfast. All at once his sight becomes dim, he finds himself very giddy; he may even faint outright, but things generally seem to revolve around him, till he is relieved by copious vomiting. Subsequent attacks may occur without, as Trousseau thinks, any discoverable indigestion or excess of diet.[1]

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[1] It is doubtful indeed whether the stomach is at all the seat of the real lesion in these cases. Thus Trousseau remarks, "I have frequently asked myself whether the treatment which apparently affected the digestive organs was not, unknown to me, addressed to the nervous system."

The vertigo is even more sudden, violent, and intermittent than in Ménière's disease, and the prostration great. If there is no indigestion, what causes the later attacks? Now it happens that vertigo occurs often in migraine, and though it is here usually referred to preceding sight and touch-affections, the migraine itself is frequently defined as caused by vasomotor changes (shown by the turgid arteries, &c.) which are produced by so called nerve-storms. Migraine, says Bristowe, has a close pathological relation to epilepsy, and, I think we may add, it is closely related to certain vertigoes.

To look at it in another light. Besides the suggestion of Trousseau that this stomachal vertigo is purely a nervous affection, there are other facts pointing to some intimate connection between the cerebellum and the stomach. Cyon, by injuries to the canals, produced vomiting; so too it exists in cerebellar or Ménière's disease: irregular movements cause sea-sickness: stimulation of cerebellum (Schiff) causes peristalsis; in typhoid there is again notable vertigo; and lastly, Ferrier oddly remarks, that in the felidæ, where equilibration is singularly perfect, there is a great development of Pacinian corpuscles in the mesentery. Whether we accept Woakes's or H. Jackson's theories, the number and variety of irritations in the vagus area with which vertigo occurs is startling.

Laryngeal irritation in bronchitis and emphysema, and even from polypi, is, according to Dr. Gee, a powerful excitor of vertigo. The patient's face is livid and turgid; he becomes giddy 15 or 16 times a day, and presents a convulsive movement of the arms, which recalls their discoloration in aural disease. Can there be a vasomotor change affecting the canals? clearly not through the inferior cervical ganglion. (*a*) Charcot suggests the superior laryngeal. The vagus itself being in close relation with the lower peduncle, may transmit through it a current to the equilibrium centre. (*b*) Or if we remember that irritation may spread up the mucous membrane to the Eustachian tube, the case becomes an aural vertigo. (*c*) Lastly, the sympathetic branch from larynx passes to the superior cervical ganglion, which gives off a branch to the external carotid, explaining the turgidity of face, and others through the carotid plexus to tympanic plexus and fenestræ, while the tensor tympani is reached by the small petrosal.

In *influenza*, and perhaps in mumps, we find exceptionally great tendencies to vertigo, and these are just the two diseases in which the invasions of the Eustachian tube are very common. Here again we need not ascribe it to general disorders of the brain-circulation, but to hyperæmia of the middle ear.

Anæmia is one of the most common antecedents of giddiness. Not only is loss of blood a cause, but various morbid conditions which are (from their frequency) perhaps more interesting to the physician than the local maladies we have been considering. To my mind this giddiness is a most mysterious matter, though we are apt to think of it as only the confusion arising from weakness. Yet why should the man who is suffering from anæmia feel himself or his surroundings *move* in either direction? It is very different from the faintness or confusion that flow from his weakness. We have seen reason to associate vertigo in many cases with an irregular outpour of energy from the cerebellum in certain directions. Can the results of anæmia be thus explained? (1.) In most cases of anæmia there is an alteration of the pressure of the blood. This can act through the canals on the cerebellum, just as tapping them does. (2.) As the deficient or abnormal blood causes a greater tendency in the nerve cells to explode, we get convulsions and failure to maintain equilibrium, because the efforts or discharges are now unmeasured and irregular. In either case we are acting on *the organ* of co-ordination, and produce in it a rudimentary and imperfect action, which is reflected in consciousness as vertigo, or a special form of motion.

Not only in chlorosis, and pernicious anæmia, hæmorrhages, amenorrhœa, menorrhagia, and heart-disease does it occur, but also on leaving a chamber of compressed air, or after the sudden loss of cerebro-spinal fluid.[1] But here we meet with a difficulty. Why does not vertigo occur every time we fall asleep, since the brain becomes anæmic then? The diminished blood then clearly does not cause explosions more numerous than usual. We die our daily death without a moment's giddiness, so that *changes in the circulation of the brain itself cannot be of much importance*, although those in the general circulation are. Thus we are again led to the stimulation, not of the whole brain, but of some end organ, possibly the canals.[2]

The action of therapeutic agents and poisons agrees singularly well with these results. The depressants are remarkably apt to cause giddiness, and by these, as Dr. Fothergill says, the tone of the arterioles is so relaxed, that the patient is actually bled into his own body. Then, again, nitrite of amyl, though uncertain in its effects on different people, very often causes giddiness by this relaxation of vessels. In opium, alcohol and carbolic acid, both results are produced; but there may be a doubt whether the effects of belladonna are not produced indirectly through distortion of vision. There is some evidence for the opposite view, such as the flushing of the facial capillaries. The dryness of the throat may point to pneumogastric changes. The giddiness of tobacco-poisoning is not less marked than that of what Woakes calls its antidote, camphor; and if we examine Böhm's classification of drugs affecting the circulation, we get a pretty complete list of those causing vertigo.[3] Now there is one drug, chloral, which Ringer and others never mention as causing it, nor do I recollect an instance myself, and yet it at once causes anæmia of the brain. This perplexed me a good deal at first; but on referring to the experiments of Bouchut, Andrews and Anstie, we find that in ordinary doses the pulse is not affected, and that, if anything, there is no fall, but a slight initial rise of blood-pressure, and even this is not beyond a doubt. That is, in a clear case of change in cerebral circulation there is no vertigo. Dr. Moxon has given strong reason for thinking that very few of the changes in the general circulation, which are frequent causes of giddiness, affect the cerebral.[1] The most constant symptoms of an overdose of gelsemium are giddiness and dimness of sight, with little or no effect on the blood-pressure, the mode of action probably being through injury to the optic centres which are in communication with the cerebellum. The vertigo is increased by motion, and is apparently subjective, *i.e.* of the patient going round, as in cerebellar lesions. This, I think, affords a contrast to poisons acting on circulation, and to excentric vertigo generally, and Ringer finds it gives relief in Ménière's disease.[2]

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[1] Thus Magendie produced staggering and apparent giddiness in a dog by puncture and withdrawal of the arachnoid fluid.

[2] But will not sleep-anæmia entail stimulation of the canals? Not of necessity, for there is no evidence, as far as I am aware, showing that the arterial contraction extends to the auditory artery, and if it did, the simultaneous quieting of the cerebellum would counterbalance any message from the canals to it.

[3] Böhm's list (abridged), 'Brit. Med. Jour.,' January, 1881. "Drugs acting on circulation:"

1. Those acting on heart direct, and when removed from body, causing increased energy; *e.g.* digitalis, helleborein, veratria, physostigma, camphor.
2. Those acting on heart direct, and when removed from body, causing enfeeblement; *e.g.* copper, zinc, tartar emetic, apomorphia (?)

In chronic alcoholism a rare form of vertigo appears, in which objects seem turned upside-down. This has been also observed in gout. In ordinary drunkenness there is paralysis of vasomotor centres and of the muscular sense, the vertigo being generally objective.[3]

In Ringer's list of diseases, where high tension is present, almost

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3. Excitors of (*a*) automatic centres, (*b*) inhibitory ganglia; *e.g.* (*a*) alcohol, chloroform, chloral cyanogen; (*b*) muscarin, nicotin.
4. Excitors of both; *e.g.* atropia, and (?) nicotin.
5. Stimulants of cerebral centres of cardiac nerves; *e.g.* prussic acid, amylnitrite, CO, irritating gas in throat.
6. Stimulants of walls of vessels; *e.g.* tannin and astringents generally.
7. Stimulants of vasomotor centres; *e.g.* (?) ammonia, strychnia, picrotoxin.
8. Paralysis of vasomotor centres; *e.g.* ether, chloroform, chloral, alcohol, aconitia and irritating gas.

[1] He lays stress on a self-regulating mechanism, by which the veins are locked when the supply is reduced; on another, by which the turgid choroid plexus presses laterally on the vagus trunk, and on the flow in and out of the cerebro-spinal fluid. He shows that enormous atmospheric pressure has no effect on the brain, and that epileptic convulsions cannot depend on brain-anæmia produced by vasomotor changes; for while these only reduce the size of a vessel ¼, and in the long time of from 4–18 seconds, Kussmaul could never produce convulsions, if even one out of the four arteries to the head were untied. It is true he regards vertigo as caused by changes in the cerebral circulation, but his arguments on convulsions really support my view that vertigo cannot be so caused.

[2] For comparison with Böhm, I subjoin the chief poisons causing giddiness, as given by Guy and Ferrier.

Narcotics: opium and morphia. *Deliriants:* Belladonna and camphor, &c. *Inebriants:* alcohol, carbolic acid, nitrite of amyl. *Convulsives:* (?) strychnia. *Heart depressants:* tobacco and lobelia. *Heart asthenics:* HCy, aconite, digitalis, colchicum. *Asphyxiants:* CO, CO_2, and H_2S.

[3] Anstie gives as the order of phenomena: The vessels are contracted, then relaxed, with paralysis of the vasomotor centres and cord and loss of muscular sense. Intellectual confusion, paralysis of respiratory centre, and lastly of heart follow. The blood is charged with alcohol, which stops oxygenation, and the nerve substance is paralysed, for when a living nerve is surrounded by alcohol it ceases to conduct.

every one is marked by the presence of vertigo; in degeneration of vessels, kidney disease, gout, jaundice, lead-poisoning, ergot and gallic-acid poisoning, fevers, &c., this is the case. There seems to be only one common property in these widely different conditions, viz. change of blood-pressure, which would readily act on a cerebral artery where there is a bony channel, together with hardly any anastomosis, and an end-organ easily stimulated by pressure.

In fevers there is often an accumulation of fluid in the middle ear, which requires no explanation.

The action of quinine and salicine in producing giddiness, though well marked, is so little understood, that I can make no use of it.[1]

In epilepsy there are two varieties of vertigo. In the *grand mal*, 77 per cent. suffer from prodromal attacks of it. This is always of falling towards one side,—never, as in brain-disease, of spinning round, nor as in excentric vertigo, of the room moving. The *petit mal* frequently consists solely of attacks of giddiness. Besides these cases, epilepsy is often brought on by ear-disease, and then paroxysms of vertigo are a marked feature.

How is it caused? Dr. Moxon speaks of it as produced by the momentary stoppage of the heart, which he thinks occurs in each fit. This might act by radiation from vagus to 7th or (through altered pressure) on the canals. But the vertigo occurs often at a very early stage, and must therefore precede heart changes. Thus, as to the brain anæmia said to exist, Bristowe argues that it may be a co-effect and not the cause of the tonic convulsions. In the general character of this vertigo there is reason to think it differs from the last class, and is caused either (1) by altered sensations, or (2) if, with Reynolds and others, we think that the beginning of an attack consists in a sudden explosion of nerve-cells affecting the medulla, and causing convulsions inhibiting the heart's action, then the early giddiness would be merely an extension of this wave of disturbance to the cerebellum and auditory nerve.[2]

In sunstroke, vertigo occurs in one only of the three forms. It is not met with in the syncopal or asphyxial types, according to some authorities, but only in the hyperpyrexial, which is also marked by epileptoid convulsions.

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[1] On the one hand it is asserted by Kirchner that quinine produces congestion of the labyrinth; but, as Dr. Moxon has shown, post-mortem appearances are very deceptive in these matters. Others again consider it causes anæmia. Moreover, Woakes was led to discover that hydrobromic acid cures the vertigo of labyrinthine congestion by an observation of Dr. Fothergill that it prevents the aural troubles of quinine. This, of course, supports Kirchner's doctrine.

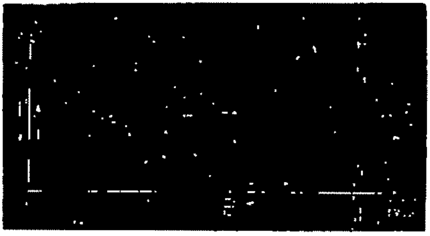
[2] It has been objected that if vertigo is, as I suppose, an affection of one organ, the cerebellum, in every case, why is it sometimes the sole symptom of epilepsy for long periods, alternating with rare attacks of the grand mal, and in other cases similar vertigo has no connection with epiplepsy whatever? Even when no other symptom is visible, there is probably in the epileptic vertigo an unperceived epileptic change. This is the foundation of the whole, and its effects may overflow the organ of equilibration or not; it has this alternative loop to traverse, but this loop or organ is equally affected by non-epileptic causes.

In locomotor ataxy we have a failure of co-ordination rather than of equilibration, and though these react on each other, so that loss of motor co-ordination renders equilibration impossible, or *vice versâ*, still a distinction can be made. As loss of equilibration alone is accompanied with vertigo, the latter affection is by no means so frequent in tabes dorsalis as might be expected. In an analogous affection, a man whose leg is "gone to sleep," as it is called, may stagger without any vertigo.

It will, however, be useful to consider this parallel disturbance of co-ordination. While the centres for real co-ordination are in the mesocephalon, the apparatus for combining a few muscles together for one act lies to some extent in the cord. Both may be involved. There has been a very great discussion whether the sensory part of the mechanism is at fault, or the central, or the efferent.

To me it seems that Erb's view is satisfactory, that it is a motor affection, that the co-ordinated stimuli sent down to maintain any complete act are cut off in their descent by lesions in certain unknown tracts in the cord, and that the inco-ordination cannot be accounted for by the anæsthesia, though it may be increased by it. In the Spaeth Schüppel case,[1] complete spinal anæsthesia existed without ataxia: the man walked, ate, and dressed himself, as long as his eyes were open, perfectly. In other cases complete ataxia existed without any anæsthesia. The often discussed tottering in the dark depends on cutting off the sensory impressions which might assist conscious co-ordination.[2] The essence of the disease is failure to co-ordinate and not failure of sensation.

When vertigo exists, it arises from (1) the accompanying diplopia, strabismus, and (2) the conflict of sensations produced by partial anæsthesia. Generally, though there is inability to co-ordinate for movement, there is power to maintain equilibrium, as far as the unmanageable limbs can be governed; there is a determination of what is needed, but not power to move the limbs as desired. We may represent these results diagrammatically, thus:—



A. Centres of consciousness (sensation and volition). B. Co-ordinating centres. C. Equilibrating centres. M. Muscular system. The arrows indicate that the tracts are everywhere double, and consist of both afferent and efferent fibres.

In tabes, B is cut off from the muscles, and reeling occurs. There may or may not be an affection of sensation, but there is nothing to cause giddiness in B's being cut off. Now if C be irregularly stimulated by A, or receive contradictory reports from the senses so that it acts irregularly, its effect on the centre A will be represented in consciousness by vertigo, and B will be misdirected, so that true vertiginous reeling occurs.

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[1] Ziemssen, xiii. p. 88.

[2] See "Controversy on the Theory of Locomotor Ataxy," in 'British Medical Journal,' Dec. 6, 13, 27, &c.

As we have said, reeling may occur when A is destroyed; *i.e.* C can act on B without affecting A.

Finally, it is worth noting, that if C be cut out of the system altogether, co-ordination can go on consciously by A alone, or by A and B, as in the pigeons whose cerebellum was removed.

Sight sensations must be very briefly considered, though their importance is great. The superior peduncles of the cerebellum are found to begin at the red nucleus just below the corpora quadrigemina, probably bridging the gap between the optic tract and the organ chiefly concerned with equilibration. Some authorities would trace a connection in the substance of the valve of Vieussens itself. In practice we find an immense number of vertigoes caused by eye-affections. (1) Myopia, nystagmus, paralysis of sixth or third nerve, and amblyopia are instances. G. Stewart gives a case of minor nystagmus, where the giddiness was stopped by pressure fixing the eye, when all other treatment failed. When one eye is *suddenly* destroyed, rotation takes place, because there is inequality of stimulation to the centre of equilibrium. (2) Looking down a precipice is one of the most common causes, and here the impressions received by sight contradict those from other senses; touch, the muscular sense, &c., by which we ordinarily are made aware of our equilibrium. Or we may explain it thus:[1] if from every side A B, &c., we usually receive certain visually produced sensations with corresponding motor outpour, and we are suddenly deprived of those on one side A, then our outpour of energy tends to be limited to one side B, and to drive us over in the direction of A. This, opposed by the other senses, causes giddiness; but if we give attention only to the sight of the chasm, we actually fall. If we traverse a plank over a gulf, there is giddiness, but none if the plank be on the ground. Waltzing, stopping after waltzing, or looking at others doing so, equally cause it by the comparatively slow motion of the eye failing to keep up with the object, and then lapsing into irregularity. In sea-sickness there is disagreement between the eye, the touch, the muscular sense and the sense of equilibrium, or, Cyon says, between our perceptions at the time, and our standing representation of ideal space obtained through the canals. In the *Agoraphobia* of Westphal and others, the patients who usually guide themselves by the vertical lines of neighbouring things, break down in crossing open spaces. There is not actual giddiness, because, perhaps, no conflict between senses, only an absence of direction from one.

Equilibrium thus depends on the agreement of the sensations from both our own bodies, and from surrounding objects, and as soon as the sensations disagree, vertigo follows.[1] Revived mental states are a still more interesting cause, but I have no time to discuss them.

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[1] Others explain this again by saying that we are unable to measure the depth of the chasm, and this lack of measurement causes the loss of equilibration.

In conclusion, I have tried to examine some of the typical cases of vertigo, to inquire *whether it be a perversion of some normal function*, and we have seen that it can generally be referred to a consciousness of disproportioned attempts to maintain equilibrium, there having been in our cases either such attempts, or processes associated with them, or affecting the machinery for carrying them out.

This mechanism most probably includes the cerebellum, acted on by impressions, mental, visual, labyrinthine, tactile and muscular, obtained through various end organs. It has seemed needless to refer such a definite and elaborate sensation to changes in the brain-circulation only when there are definite organs able to produce vertigo upon stimulation; and without accepting as completely proved any theory of the canals, or Dr. Woakes's theory, there is strong reason to refer most of the excentric vertigo to processes acting through them on the cerebellum. In what single particular is any stomachal or toxic vertigo different from that of Ménière's disease confessedly acting through them?

At the same time I am aware of the danger of forgetting the plurality of causes, and of reducing the infinite variety of nature to the meagre uniformity of the human mind. The difficulties of getting evidence on a subjective symptom such as this are great; but so far as I can judge, there are two types; the subjective, where the person himself seems to move, produced by direct injuries to the cerebellum, epilepsy, &c.; and the objective, which includes aural stomachal varieties, and those caused by rotation, diseases of touch or sight, or affecting the circulation.

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[1] As Wundt (Physiologie) remarks, so soon as the sensations which relate to space conditions are altered, vertigo (Schwindel) occurs, because all our volitions with regard to a composite action are strictly proportioned in each elemental part, though we placed before our consciousness only the attainment of a general aim.

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Sleep-Walking and Hypnotism. By D. Hack Tuke, M.D., LL.D., F.R.C.P. London, Churchill, 1884. 1 vol. 8vo. pp. 120.

Dr. Hack Tuke has done admirable service by associating the subject of sleep-walking with that of hypnotism and the so-called mesmeric state, for by so doing he has shown the common ground on which all these conditions depend, and has employed a method which affords a clue for unravelling the various phenomena exhibited under these different names. In all these peculiar manifestations of the human mind the essential feature is the partial or total loss of the ordinary volitional or guiding power, and the predominance of an instinctive or automatic form of action, in which there is often a complete subjugation of the faculties to the will of another person. At the present day the question awaiting solution refers to the actual state of the brain, and the nature of the mental processes which take place during these manifestations, and not to the mode in which they are brought about, for it would seem that from a variety of causes this automaton-like action may be displayed, and may therefore be regarded as one which is natural and common to all human beings. Probably every one under the rightly favouring circumstances might be thrown into this trance-like state, and therefore it is a phase which is absolutely necessary to be examined by those who make a study of the human mind. Formerly the automaton condition of which we speak was thought to be wholly abnormal or peculiar, and was to be induced only by some extraneous influence, such as the passage of the hand over the body of another person, through whom some special magnetic power was transmitted. This magnetic influence was supposed to pass from one person to another, and the object was said to be mesmerized; Mesmer being one of the earliest experimenters. When Braid found that any individual by gazing steadfastly at a bright object might fall into this trance-like state, it was obvious that no theory of animal magnetism was required to account for it, and now we are able to declare that even this simple process is not essential to produce it, for some persons may fall into this hypnotic condition without the use of any extraneous means whatever; and moreover, that somnambulism seems in every way identical with it. Dr. Tuke has therefore done well to insist on a study of the phenomena of sleep-walking in connection with that of mesmerism or hypnotism, and to ask the assistance of others in making accurate observations of all cases of somnambulism. In the case of a girl, which Dr. Tuke reports, whether she fell into a state of somnambulism, or was thrown into the hypnotic state by another person, she displayed exactly the same phenomena under these different circumstances.

Probably modifications of the hypnotic state, where there are partial losses of consciousness, and yet various bodily actions continue, are common enough. Every one can relate in his own person of the performance of certain acts whilst he was altogether unconscious of them. In concussion of the brain, in drunkenness, and more especially in epilepsy, such temporary loss of consciousness is not uncommon. For example, the writer is acquainted with a medical gentleman who has had fits at long intervals. After one of these he found that he had lost his memory of acts which he had immediately before performed, and he in consequence requested his wife to accompany him on his rounds and take notes of all that he did. After visiting a patient and returning to his brougham, he made his wife write down the prescription from his dictation, and she would then direct him to the next place. The same process was repeated, until he had finished his rounds. When he arrived home he had no recollection whatever of the occurrences of the day. This forgetful state continued for three days, when his perfect consciousness returned. These circumstances were related by himself in the presence of his wife, who confirmed them. Moreover he never subsequently heard from his patients that any peculiarity had been observed in his manner during these three dreamlike days. In these cases, as in those of somnambulism and hypnotism, the expression "loss of consciousness" is not altogether accurate, for the subjects of these states reciprocate to all that is said or done, and their senses are often very acute. A state of unconsciousness is one of coma, where a patient cannot be roused, and his senses are dead; whereas in the hypnotic or mesmeric condition the perceptive centres are still active, and the senses open to impressions, but the highest centre associated with clear mental consciousness appears to be dormant, and is not roused into activity by the ordinary stimulants. A condition somewhat similar is seen after concussion of the brain: for example, a man receives a violent blow and is rendered insensible; after a time he partially recovers and then acts like an automaton, he may walk about his room, dress himself, and perform other acts to which he is accustomed, but of all these he will not have the slightest recollection some days afterwards. There is thus apparently a lesser degree of the automaton state, that in which the person acts as if he were conscious, and yet is found to be soon afterwards quite oblivious of the scene in which he took part. For example, a man may in a drunken brawl resent insulting words, and be quite conscious of receiving a blow from another, and be able to retaliate; yet on the following morning he has no recollection of the fray, and the only proof to him of the fact is the loss of teeth, of which he was until then quite unaware.

In removing from the subject of trance or hypnotism the various causes which may induce it, we get rid of any discussion respecting mesmerism, animal magnetism, or other influence; we place it on a broader and simpler basis. We regard it as a normal condition, very common in a modified form, and only in extreme instances to be regarded as remarkable, as when seen in somnambulism, trance, or hystero-epilepsy. It is very probable that many of the death-like conditions into which animals may be thrown are of a like nature. At a lecture lately delivered by Mr. Langley at the Royal Institution, he had no difficulty in rendering a pigeon, a frog and an alligator perfectly motionless and perhaps unconscious. What we want to ascertain is what portions of the brain are in abeyance during this state, and which are active; also whether some portions are in a state of excessive activity, as for example those of the special senses. It is certain that the retina answers to impressions; for the somnambulist avoids obstacles, and can walk along the most dangerous paths, and yet the impression is not carried deeper into the sensorium, for some particular friend might be avoided as an obstacle, but not recognised. It is interesting to note modifications of this hypnotic or sleepless state as constantly occurring in ordinary life, for we thus remove it from the region of mystery and place it amongst the normal phenomena of the human mind. For example, the writer having been away from home for some time, returned to London, when on passing along a street he observed his father crossing at the end of it. The latter looked straight at him and walked on. Subsequently, when they met at home, the father said to the son, "I have seen you before to-day, but I do not know where." It is clear that his mind was preoccupied, and therefore received only the faintest impression of the image thrown on the retina, and this was not strong enough to arrest his progress. The various cases related by Dr. Tuke exhibit the condition of the senses in the somnambulistic state, in various degrees of activity, and thus although in some cases they are highly sensitive, in others they appear to be not in action, and as if the body was not so much influenced by them as by some powers within. For example, a hypnotic person whilst writing on a piece of paper would still continue to write on, even when a screen were placed before the eyes. This resembles what is sometimes seen in an epileptic, who will continue some mechanical movement after the fit has seized him, and he is quite unconscious.

The most remarkable phenomena which have to be studied in these cases are the acts which are due to suggestion. The subject of them appears to be like a pliant piece of machinery, ready to be acted upon or played upon by any extraneous force. He will apparently do anything he is bid, or believe anything which is told him Many of these phenomena are explicable on the ordinary theory of reflex action which accounts for a large part of our thoughts and sayings in the normal state; but there is one phenomenon which, if correctly observed, is most remarkable, and gives some sort of warrant for the old belief in animal magnetism. This is the influence which the operator has over the subject by merely moving his hand when he stands behind the latter. It is credibly related that if the operator be behind him or her, and move his hand, the patient will approach or retreat as he beckons or warns off. If this is to be explained by the influence of the ordinary senses, it implies that the cutaneous surface of the patient is wrought up into a state of sensibility far beyond what is known in the normal condition, so that it becomes cognizant of vibrations of the air made at long distances. It was maintained by the late Dr. Beard, of America, that in hypnotism the perceptive centres and some other portions of the brain were in a state of hypersensibility, and he illustrated his meaning by pointing to a chandelier having a number of gas-burners. After comparing the full-lighted chandelier to the active brain, and the same when extinguished to the torpid brain, as in sleep, he took the case where one or two burners only were alight, and remarked the great height of the flames in consequence of their being supplied with a superabundance of gas whilst the others were not in use. This example he compared to the hypnotic state, where one or two of the senses were in a state of inordinate activity, whilst the other perceptive centres were dormant.

The whole subject is one of paramount interest in reference to the philosophy of the human mind, for it shows that consciousness is not an essential element in all our mental acts. It is now generally admitted that the higher centres of the brain may be in full operation without consciousness being called up, and to this the term "unconscious cerebration" is given. During sleep many instances might be related of the most complex mental operations being performed, and the subject of them being quite unconscious. He has only become aware of the recent activity of his brain upon waking up to the results in the morning. Although, under ordinary conditions, our mental state associated with consciousness directs our acts, we must admit that much must go on within us without consciousness or volition on our part. If this be the normal state, those of which we have been speaking, known as hypnotism or somnambulism, is a mere exaggeration of it; and if, as is generally admitted, reflex phenomena are everywhere apparent in mental as well as in physical states, we see that in hypnotism there is merely an extension or excess of a normal process. It can be easily understood how, the inhibitory actions of the will being removed, the brain can be more easily acted on through the senses.

Unless one has directed his attention to the fact, he is not aware of how large a part of our conversation in ordinary life is due to reflex action. The more a person can keep to the point of his argument, the greater his intellectual power; and, on the contrary, the being led away by new words or new thoughts is the very picture of weakness, and is little more than a representation of reflex action. All writers have discerned this, from Shakspeare to Dickens, in describing the speeches and conversation of illiterate persons. Albert Smith was in the habit of giving a sailor's yarn, in which one idea suggested another, and this a third, until the story went on without any single idea running through it, and in which the starting-thought was totally lost. In a less degree, in ordinary table-talk, the subject started for conversation is often altogether forgotten; words or names let fall suggest some new thoughts, these when expressed suggest new ideas to others, until the company seem little better than automata. The more the subject is examined, the more intimately we shall find our thoughts and actions due to reflex action, so that we sometimes "answer a question without a thought," and for the time being are acting the part of the somnambulist. Dr. Tuke has done wisely by bringing together a number of allied phenomena, and showing how they are parts of the physiological law; how in various degrees, the guiding or inhibitory action of the will being removed, the lower functions of the brain can be set in operation through the senses.

Dr. Tuke's book naturally touches upon subjects of great practical importance, which are an outcome of these exceptional mental conditions. For example, the responsibility of the subject for acts done in the somnambulistic state, a question of the gravest importance, and one which had had to be discussed in tribunals of law. The following case told to the writer by a military friend might have had a very tragic result. When young, he had occasionally walked in his sleep, and whilst in camp in India, he one night found himself in the open country with nothing on but his nightshirt. He at once realised what had happened, looked about him to discover where he was, and hurriedly returned to his quarters. On entering the camp, the sentinel on duty presented arms after the usual fashion to an officer, and let him pass. It then became evident that the same formalities must have been gone through when he made his exit; he thought it prudent, however, to say nothing of his adventure, and therefore made no enquiries of the guard. There can be no doubt that if a private soldier had been seen walking out of barracks in a similar manner it might have fared very differently with him. We strongly recommend Dr. Tuke's book for perusal, for, although small, it contains facts of a most valuable and interesting kind, which must be thoroughly examined before a complete science of the human mind can be attempted.

S. WILKS.

A Text Book of Human Physiology. By DR. L. LANDOIS, Professor of Physiology in the University of Greifswald. Translated from the fourth German edition by WILLIAM STIRLING, M.D., Sc.D., Professor of Physiology in the University of Aberdeen. Vol. I. 8vo., pp. 514. London: Charles Griffin & Co.

PROFESSOR LANDOIS' work on Physiology is particularly distinguished by its practical nature and by the constancy with which the Author brings the facts of Physiology into relationship with medicine. It is a book written especially for medical students and medical practitioners, and the success with which the Author has adapted it to their wants is shown by the fact that it has already passed through four large editions in four years. Each section begins by giving a full description of the normal processes of life, and this is followed by a short account of their pathological variations, and a brief notice of the ways in which they are affected by various drugs. The work is thus calculated to direct the attention of the student towards a rational system of treatment, and to help the practitioner rightly to understand and treat the cases under his care. With this object in view, all the methods of investigation which may advantageously be used in practice are fully described, and more space is given to histology than is usual in text books of physiology. Professor Landois seems to have a special faculty for diagrammatic illustration, and his schematic drawings render the appreciation of histological and anatomical facts much easier. As illustrations of these we may mention his schematic drawings of the air-vesicles of the lung (Fig. 99), of the gastric mucous membrane (Fig. 136), and of a lobule of the liver (Fig. 141).

Professor Stirling has translated the work well, although here and there we notice a few imperfections. The most striking is his translation of the term "Selbst-steuerung," applied by Brücke to the action of the heart in producing ventricular dilatation as a consequence to ventricular contraction. Brücke supposed that during the ventricular systole the semi-lunar valves covered the opening of the coronary arteries, so that blood could not enter into them, but during ventricular relaxation the valves left the coronary openings free, so that the blood flowing from the tense aorta into the walls of the ventricle would tend to dilate it actively, and thus fill it more completely with blood for the succeeding systole. This action is like that of the valve which is attached to the cylinder of a steam-engine, and causes the movements of the piston to alternate. To this kind of gearing the term "Steuerung" is applied; but Dr. Sterling, instead of translating "Selbst-steuerung" as "valve-gearing," or "automatic regulation," or "automatic valve-regulation," or something of the kind, has translated it "Self-steering." No doubt the word employed by Brücke sometimes has the meaning given to it by the translator, but this does not seem to us to be at all appropriate to the action of the heart. The translation in general is, however, very good, and freer from German idioms than such translations usually are.

The work is, however, not a mere translation.—Dr. Stirling has made large and valuable additions to it. In places where the German edition begins abruptly, and seems to assume an amount of knowledge which the student may not possess, Dr. Stirling has supplied the necessary introduction; he has filled up any lacunæ that were present either in the histological descriptions or accounts of the functions of the various organs, and he has added descriptions of several methods of investigation omitted in the German work. The present volume contains the physiology of the blood, of the circulation, of digestion, of absorption, of animal heat, and of tissue metabolism. Dr. Stirling has added 70 woodcuts to the 106 contained in the German edition—and has thus still further added to the value of Professor Landois' work, which itself is one of the best and most practical treatises on Physiology we have ever seen.

T. LAUDER BRUNTON.

A Treatise on the Theory and Practice of Medicine.—By JOHN SYER BRISTOWE, M.D., F.R.S., &c. London: Smith, Elder, & Co., 1884. 1 vol. 8vo., pp. 1240.

Lehrbuch der Speciellen Pathologie und Therapie der inneren Krankheiten. By ADOLF STRÜMPELL, M.D., Professor of Medicine in the University of Leipzig. Leipzig: Vogel, 1883-1884. 2 vols., 8vo.

DR. BRISTOWE's treatise is so well known that it is unnecessary on our part to do more than welcome the fifth edition, and congratulate the author upon his well-deserved success. The part relating to nervous diseases occupies about 250 pages, and has from the beginning been a distinguishing feature of the book, which was the first of its kind in England to offer to the student a clear and reliable digest of recent neuropathology. Dr. Bristowe has completed his survey by adding a chapter on Insanity, including general paralysis, a subject of the highest importance for the general practitioner, as well as the student of the nervous system and its ailments, but which is still too often relegated to special treatises.

Professor Strümpell's textbook is a larger work than Dr. Bristowe's; but from the reception it has had in Germany at the hands of both students and practitioners, it bids fair to replace the old favourite volumes of Niemeyer. From the author's reputation as a neurologist, it was to be expected that the portion of the book relating to nervous diseases should be worked up with special care and success. Our anticipations have not been disappointed; and the first part of the second volume, which forms a distinct whole and contains 440 pages, gives us a most accurate and fairly full *résumé* of what is known on the subject it discusses. About 60 illustrations assist the reading of the text, which is written in a style, the lucidity of which many of the author's countrymen would do well to imitate.

We shall not attempt to criticise in detail the various chapters of the book, especially as we know that a second revised edition is now occupying the author. But we wish to draw the attention of the reader to the view taken by him of certain acute inflammatory processes. In a lecture published about a year ago,[1] Prof. Strümpell has given a clear account of the ætiology of systematic nervous diseases. He mentions and discusses four orders of causes: embryonic predisposition, over-use, psychical excitation, and toxic influences, among which "germs" play an important part. Acute poliomyelitis, acute multiple neuritis, and a form of acute hydrocephalus, are instances of nervous diseases of infectious nature, according to the author. Even those who are not prepared to go the full length with him will not fail to recognise the importance of the subject. As Professor Grasset says in his article on the relationships between hysteria and the tubercular diathesis,[1] our modern neuropathology has hitherto been absorbed in the study of lesions, and in the explanation of symptoms; of disease itself, the cause of those lesions, we know as yet very little. A true etiology is not only required for a complete knowledge, but is the condition of success in the treatment of nervous diseases. "Sublatâ causâ, tollitur effectus," should be the motto of our therapeutics, which are still in most cases painfully inadequate even to relieve the symptoms against which they are directed. But we need not despair, in presence of the rapid developments taken by neuropathology in the course of the last few years, and of which a most striking proof will be found in comparing the two Handbooks now before us with those in use during our own student-days.

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[1] 'Ueber die Ursachen der Erkrankungen des Nervensystems.' Leipzig: Vogel, 1884; pp. 22.

A. DE W.

L'Homme et l'Intelligence. Fragments de Physiologie et de Psychologie. By Professor CH. RICHET, Agrégé à la Faculté de Médecine de Paris. Paris: Alcan, 1884. 1 vol. 8vo. pp. 570.

Des Suggestions Hypnotiques. By Dr. A. PITRES, Professor in the Faculty of Medicine at Bordeaux. Bordeaux: Feret, 1884. 1 vol. 8vo. pp. 64.

De la Suggestion dans l'état hypnotique et dans l'état de veille. By Dr. BERNHEIM, Professor in the Faculty of Medicine of Nancy. Paris: Doin, 1884.

THOUGH very unlike in title and external appearance, we may well group these three books together. Professor Richet assembles in one volume seven essays written at different times, but having a common aim—that of studying mental phenomena in connection with their physical embodiment and their cerebral substratum. The first subject, "Pain," and "The Causes of Disgust," are treated by the method characteristic of the physio-psychological analysis. In the second, "The Causes of Disgust," the author tries to reduce the phenomena of physical and moral disgust to a common law. He argues that in the midst of all the diversities of human tastes there runs a uniform thread; by following which up to its origin we reach the common principle at the root of all. This principle is that we are led to avoid hurtful, or even useless, things; disgust therefore may be defined as being primarily an instructive feeling, the result of hereditary experiences, of self-protection; but which, by association of ideas, is liable to almost infinite extension and fluctuation.

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[1] 'BRAIN,' 1884.

The Essay on the "Poisons of Intelligence," discussing the psychical effects of alcohol, chloroform, hashish, and opium, is full of interesting, if not always new, matter. Psychology must become experimental in order to become more exact, and cease to be a mere description of subjective phenomena, in order to become truly scientific. It must also be subjected to the comparative method in two directions; seeking to unravel the complex phenomena of our own minds, first by discovering the primordial constituents of conscious life in the lower organisms; second, by observing the disintegrating effects of disease. The superiority of the experimental method lies in the fact that the conditions of observation may be altered at will, though within necessarily narrow circuits, and that the observer may apply the method of introspection by using himself as the subject.

The chapter on intellectual "Poisons," and the next on "Artificial Somnambulism," contain many illustrations of the value of the experimental method; whilst the two Essays on Hystero-epilepsy ("Démoniaques d'autrefois et d'aujourdhui,") contain psychical studies of this now familiar disease. The last study, "The King of Animals," is of a somewhat different character, and treats of the relationship of man to nature, and of his rights over the lower animals; the cause of experimental physiology and pathology finds an eloquent advocate in Professor Richet, who with much reason points out that if the utilitarian argument that animals are not to be sacrificed for the sake of gratifying our curiosity had been enforced in the case of Galvani, we should be now deprived of all the boons which the discovery of dynamic electricity has conferred upon the human race. A copious appendix of additional matter, in the shape of notes to the several subjects discussed, closes the book.

The Essay on Somnambulism—which occupies 110 pages—contains many valuable facts and views; and should be read along with the pamphlets of Professors Bernheim and Pitres by all those interested in the study of the human mind. The depth of the somnambulic state varies considerably; three stages have been described, of which the first, or preliminary, period is called that of torpor; the second, of excitation; the third, of stupor; completeness of the hypnotisation is measured by the degree of automation to which the subject has been reduced. Experiments on animals may be of use to prove the reality of the phenomena, but cannot lead to the same results as those on man, who alone is susceptible of displaying the characteristic phenomena of suggestion.

These phenomena depend upon an alteration in the normal cerebral equilibrium, and dissect, as it were, the complex manifestations of psychical life; some components of which are abolished, whilst others are placed in relief, and may even be exaggerated. The inhibitory influence of the higher centres being in abeyance, the so-called spontaneous exercise of mind is abolished; there remains nothing but certain manifestations of memory, and association of ideas strictly limited to motives supplied by the operator; the subject becomes a cerebral automaton. In this somnambulic state, also, the mutual action and reaction of mind and body upon one another are displayed in a remarkable manner; not only are certain suggested trains of thought accompanied with appropriate actions, but postures expressive of feelings, passively impressed upon the limbs, call forth the corresponding trains of thought. Under the name of "Objectivation of types," Prof. Richet describes a state in which the subject, having lost the memory of his former self, acts and speaks exactly as another person whom he is made to believe to be.

A remarkable phenomenon is that whilst the subject may lose the sense of his identity, he does not necessarily lose the memory of past events, and yields a personal reaction, as it were, to external stimuli. The whole subject of memory with reference to the hypnotic condition is one of the deepest interest. As a rule, no reminiscence of what has been going on in that state is preserved after the waking up, though it may revive during the next hypnotisation. But very important facts have been brought to light recently, showing that in sensitive subjects suggestions made to them during their somnambulism may go on acting as motives to their actions in the waking state. They then feel a blind, uncontrollable impulse to do a certain thing, possibly shameful or criminal; they struggle against the impulse, and may overcome it if the action is sufficiently repugnant to their ordinary nature, but not otherwise. The influence of the suggestion may remain dormant for a considerable period; thus Prof. Bernheim relates a case where an order given during the hypnotic condition to be fulfilled two months afterwards, was fatally executed after that interval. But we cannot enter upon the details of these and the many other interesting observations related in the works before us; we hope to have said enough to stimulate the curiosity of the reader, and make him desirous of acquainting himself with the originals.

A. DE W.

The Medical Chronicle: a monthly record of the Progress of the Medical Sciences.—Edited by JAMES NIVEN, M.A., M.B., and W. J. SINCLAIR, M.A., M.D. Manchester: J. E. Cornish.

WE heartily congratulate our Manchester *confrères* upon the excellence of the opening numbers of their new periodical. They have pitched their standard very high, and for the sake of English medical literature we hope they may sustain the effort. As the title implies, the Journal is chiefly devoted to abstracts of recent publications, which occupy about three-quarters of the entire number of 120 pages. These abstracts are carefully selected, conscientiously written, and form a solid body of readable matter, occasionally assuming the shape of short reviews or well-digested reports; and among them we may mention the admirable contributions of Dr. Ross on neurological subjects.

About thirty pages are devoted to original matter, consisting of short articles on points of practical interest. Let us note, *en passant*, that to a contribution by Dr. Railton on pseudo-hypertrophic paralysis, succeeds—an instance of *true* hypertrophy—a paper on the pulse, due to the imaginative exuberance of the ubiquitous Mortimer Granville. Otherwise the Journal does honour to the staff and their associates, among whom so much earnestness and ability is manifest. We wish them every success in a literary enterprise worthy of the rapidly rising school in the midst of which it has taken birth.

A. DE W.

Clinical Figures.—(Brain, etc., Series III.) Lebon and Co., 23, Southampton Buildings, Chancery Lane, London.

THIS third book of Diagrams of the Nervous System includes a horizontal section of the Brain through the internal capsule and basal ganglia (after Flechsig); a section through the crura cerebri at the level of the anterior pair of corpora quadrigemina (Schwalbe); one through the medulla oblongata through the middle of the olivary body (Schwalbe); and three transverse sections of the cord (after Flechsig) in the cervical, dorsal, and lumbar region respectively. They are very accurate; and, like their predecessors, will be found of great use, not only at the post-mortem table, but at the writing-desk also, for taking notes of published cases. Authors will be glad to know that Messrs. Lebon are prepared to furnish electro-plates of the figures, with lesions marked on them on the plan mentioned at page 280 of the present volume of "BRAIN."

A. DE W.

Abstracts of British and Foreign Journals.

A Case of Fatal Pemphigus-like Dermatitis, with Changes in the Nervous System.—By Dr. P. Mayer, of Strassburg. (Abstract from *Virchow's Arch.*, Part II. 1883.)—The patient was a man, æt. 65 years, and was admitted into the Strassburg hospital on November 28th, 1881. There was no neurotic family history, nor any evidence of previous disease in the patient, except that he had a double cataract.

The disease began three weeks previously with severe itching, followed by an eruption of small hard papules, which became vesicular and dried into crusts.

On admission, there was no disease of the viscera, but on the front of the chest was an eczematous-looking eruption, and on the neck some small itching papules. The eruption remained like an ordinary eczema for a week, and then pemphigus bullæ came on the trunk and limbs, with general disturbance, evening fever, frequent pulse, lithæmia, anorexia and sleeplessness, and pemphigus foliaceus gradually declared itself; large patches of cutis became discoloured, the epidermis was thrown off with very little fluid exudation underneath, and the aspect was that of a severe burn. Fresh eruption-patches came every day; but after a time bullæ ceased to form, diarrhœa set in, and he died on December 31st, seven weeks from the commencement of the eruption.

Post-mortem, there was nothing of importance in the viscera. There was a very little fluid in the dura mater spinalis, and the inner surface was pale; the anterior roots, especially in the dorsal regions, were rather thin, the cauda equina was strongly injected, the nerve fibres were rosy, the posterior roots flattened, and the cord was of normal consistency. In the neck the white substance of the posterior columns near the central canal was somewhat transparent, had a yellowish hue as far as the upper dorsal region; but here a greyish streak began on each side of the posterior longitudinal sulcus; in the posterior columns the grey substance was everywhere sunk in. In the lower dorsal region were transparent patches; in the lateral columns, more marked still in the lumbar region. There were small hæmorrhages in the galea capitis—a recent hæmorrhage on the under surface of the dura mater on the right, small hæmorrhagic-spots on the left; the pia mater was œdematous; there was nothing particular in the brain. In the cord, one centimetre below the decussation, the posterior columns were greyish-yellow.

Histological Investigation.—Small pieces of skin were put into osmic acid, and large ones into alcohol and Müller's fluid.

Microscopical examination of the skin showed simply dermatitis, with considerable cell-infiltration and loss of epidermis. The subcutaneous nerves of the fore-arm, dorsum of foot and leg, showed many empty nerve-sheaths with numerous nuclei, and scattered myeline drops; and some nerve fibres were in the more recent stages of parenchymatous degeneration, with breaking up of the medulla and accumulation of granular protoplasm, proliferation of nuclei in Schwann's sheath in many fibres, and the empty sheaths had moniliform swellings filled with myeline balls, and the protoplasm rich in nuclei; here and there completely granular cells had already formed, which in varying number clothed the empty sheaths and very fine fibres with short links, and thin myeline coverings were scattered about. Many other fine skin-nerves from various parts showed signs of degeneration, and even some of the fibres of the sciatic, and larger branches of the brachial plexus. There was nothing in the muscles or sympathetic nerves, nor in the spinal ganglia or roots. In the cord—to the naked eye there were streaks along the longitudinal fissures in the whole cervical and dorsal regions, greyish-yellow discoloured places in the posterior root bundles in the upper cervical, the transparent spots in the lateral columns of the lower dorsal and lumbar regions. Microscopically—the interstitial tissue in these parts was thickened, the nerve-tubes were surrounded by a firm connective tissue, strongly stained with carmine, but poor in nuclei, and here and there bands of fibrous tissue usually along the blood-vessels; the vessels themselves had their lumen narrowed from fibroid thickening; thickened fibrous tissue also surrounded the nerve tubes singly, and in groups near the vessels; and in parts the nerve tubes were shrunken and reduced in size, and denuded of their medullary sheath, being only visible as small coloured dots.

There were no granular cells, but only scanty corpora amylacea. The sclerosis of the columns of Goll ran in the whole length of the cord, strongest towards the posterior commissure; in Burdach's columns it was more in scattered patches about the middle between Goll's columns and posterior root zones; in the lower dorsal and upper dorsal regions there was an increase of the interstitial connective tissue in the lateral columns, though only in the neighbourhood of the posterior roots.

The anterior roots were quite healthy. In the grey substance there was only marked pigmentation of many ganglia cells, as is usual in elderly persons.

Remarks.—The changes in the skin were on the whole in favour of pemphigus, though there was more cell infiltration than usual, according to the view of some authorities, and there were too many nerve-fibres affected in the skin for the changes to be other than pathological, while they were proved not to be senile in the skin or in the cord, by comparison with the same parts in an older man.

Whilst the cord changes were undoubtedly older than the skin lesions, it is quite possible that it was only when the lesions reached a certain degree that they were of importance.

The connection between sclerosis of the posterior columns, irritation of the lateral columns and inflammation of the anterior horns, has been pointed out by many observers; but it is rare to find bullous dermatitis without previous nervous symptoms having been noticed when anatomical nerve changes were found post-mortem.

Schwimmer brings forward several cases with bullous eruption and cord lesions; his cases resembled ours, but were more severe, with the same parts, the columns of Goll, and posterior parts of the white substance affected, the process having evidently started there in the neuroglia and vascular scaffolding.

The duration of the skin disease negatived Babes's idea, that the cord lesion might be consecutive to the skin affections. Similar skin affections with cord lesions, are scattered through literature:—Jarisch's is a case in point, called by him Herpes Iris. In a woman, æt. 61, who had had a bullous eruption on the face, trunk and upper extremities for five years before admittance, and isolated bullæ here and there, and some capsules and vesicles, there were no spinal symptoms; but on her death, two months later, from pneumonia, &c., the cord was affected from the 3rd cervical to the 8th dorsal, in the anterior horns, and there was also a sclerosis of the central grey matter. Jarisch considered the whole process a neuritis in Stricker's sense. Quinquaud's cases, which developed into pityriasis rubra, also had changes in the nervous system, in the shape of a diffuse myelitis with increase of neuroglia and connective tissue round the vessels, and also extensive parenchymatous degeneration of the skin nerves.

It thus appears that, as the result of pathogenetic irritation, we may have papules and vesicles, or bullæ, or exfoliation, and although these changes are probably neurotic, coexistence is all that has been proved. It is to be remarked that the vascular changes in this case, consisting of obliteration of many of the skin vessels from endarteritis obliterans, and in the cord from a more diffuse fibrous change, was limited to those parts. It is quite possible that though this change was not recent, the nutritive changes led to sclerosis of the interstitial tissue of the cord, and when this was severe enough to compress the nerve fibres, the skin was damaged, its arteries blocked, and the process became so severe as to kill the patient. Further researches may show, whether the vascular changes in the skin and cord really possess the importance assumed by this hypothesis.

H. Radcliffe Crocker.

On Vasomotor and Secretory Troubles in Tabes.—Professor Pitres has reissued, in pamphlet form, a paper originally published in the 'Journal de Médecine de Bordeaux' (January 1884), wherein he records a series of symptoms which he observed in five cases of tabes dorsalis, and which, in the absence of other symptoms, he regards as of considerable value in forming a diagnosis. M. Strauss gave a description, in 1881, of the "Ecchymoses tabétiques," which may arise at any part of the body afflicted with lightning-pains, and the cases now recorded by M. Pitres agree with his in showing that these ecchymoses usually appear after attacks of pain, and wholly independent of any local traumatic influence. In themselves they are usually painless, are marked by much sanguinolent effusion, and occupy sites rather below the points of greatest severity of the lightning-pains, to which, indeed, they seem to bear some very definite relation. They rapidly disappear, and leave only a slight yellowish stain for a few days. In one of his cases which presented many of the more ordinary symptoms of tabes, the patient was subject to profuse sweating of the feet and lower part of the legs. This sweating was in fact the first symptom which, associated with painful tinglings of the feet, brought him under medical treatment. It was rapidly followed by impairment of vision and well-marked ataxy, and after lasting six weeks suddenly disappeared, and gave place to violent lightning-pains in the same parts. He got worse during the next twelve months, and when seen by M. Pitres in June 1883, the skin of his feet and lower third of his legs was of a rose colour, which sometimes was found universally distributed, sometimes appeared only in patches or was occasionally absent, over these parts. The same area was covered continuously with small drops of sweat, which could be seen in course of formation, although the feet at the same time felt unduly cold. The nails of the great toes were crumpled and deformed. The fact that this local sweating appeared at times when the affected area presented no difference in colour from other parts which were perfectly dry, seems to show that the condition was wholly independent of any vascular disturbance, and to support the view that the vascular and secretory functions are not under precisely the same nerve control. Cases of this kind are by no means uncommon, and the symptom sweating bears a strong resemblance to those of increased saliva flow, vomiting and diarrhœa, which have been described by several writers as met with in the course of tabes dorsalis.

M. Pitres records an interesting example of *sialorrhœa* which occurred in crises, was ushered in by a strange sensation of burning on the top of the tongue, and lasted some two or three hours. The flow of saliva was enormous, had no obvious cause, and was unamenable to treatment. These cases are all brought together with the object of showing that certain symptoms of a rare and unusual kind may appear at the very beginning of the disease, before lightning-pains, gastric or other crises, or even ataxy have become established, and that their recognition is of great clinical importance. A case of the same character is also recorded in detail by MM. Raymond and Artaud ('Revue de Médecine,' No. 5, 1884, p. 414), where local sweating of the most remarkable nature occurred in the course of tabes. The authors give a short *résumé* of the various cases already published, by Nitzelandel, Eulenburg and Guttmann, Remak, Putnam and others, where symptoms were noted as being apparently due to disturbance of the great sympathetic. All of them have these common characteristics, sudden onset of the symptom, and great abundance of the secretion, whether it be from the salivary or sweat glands, or from the stomach or intestine. In their own case a soldier began at the age of 35 to have severe lightning-pains, which were followed in the next decade by many of the more usual symptoms, occasionally by ecchymoses, by severe neuralgia in the distribution of the 5th nerve, by loss of teeth, squinting and impaired sight. He suffered when the authors saw him from severe lightning-pains in the legs, and each attack was accompanied with copious sweating from the affected limbs. Now and then, also, he has the *taches ecchymotiques.* Severe pains are also felt in the upper limbs, but more especially are they severe in the occipital and temporal regions, with giddiness and the sensation of a bar of iron piercing from one ear to the other. He has lost most of his teeth, and some of his nails are grievously altered. The most extraordinary feature in the case, however, is profuse sweating, limited to the right half of the head after each meal. About ten or fifteen minutes after eating he feels a sensation of pricking or warmth on the right side of the face, which forthwith is bedewed with limpid acid sweat which pours off in innumerable drops. There is little if any change to be noted in the colour of the right face at this time, its temperature is but very slightly higher than that of the left, and there is no alteration in the size of the pupil. The sweating occurs on the right side of the head and right shoulders, as well as on the face, but while it comes there only after lightning-pains, on the face it is after every meal. There is no defect or change of taste or smell, but the man believes there is increased flow of saliva on the right side while sweating lasts. The result of a subcutaneous injection of nitrate of pilocarpine was not different on the two sides. This symptom would appear to depend, say the authors, on some disturbance of function of the great sympathetic, in connection with disease of the spinal cord, and it is well known that degeneration is frequently found in those parts of the cord where M. Pierret, by recent researches, has placed the central origin of the sympathetic. The authors, however, say nothing about peripheral neuritis or degeneration, and it seems quite possible that many, if not all, of these strange symptoms may be due to peripheral disease of nerve-trunks, rather than to irritation of more central parts.

Féré and Others on the "Pied Tabétique."—We have before us no fewer than three papers on that rare form of arthropathy to which the name of "pied tabétique" has been given by MM. Charcot and Féré, in a description of the then known cases in the 'Arch. de Neurologie' (1883, t. vi. p. 305). Four cases were there recorded, and to them may now be added three more, possessing certain common features. M. Féré's more recent case, ('Revue de Médecine,' 1884, No. 6, p. 473), is that of a man aged

52, who came under observation in January of the present year, and who as long ago as 1872, after a severe fall, began to complain of violent pains having all the characteristics of the lightning-pains of tabes. At times also he had partial paralysis of the bladder with almost total impotence, and during the last two years occasional attacks of apparently causeless diarrhœa. There had, however, been little or no difficulty in walking, although when first seen there was unquestionable ataxy in gait, which with various other symptoms made the diagnosis certain. About two years previously his right foot had begun to swell without obvious cause, and changes in it had gradually supervened, so that when examined by M. Féré it was found thickened along the inner border, which had sunk lower than normal. This result is seen very strikingly in the impressions of the two feet, as shown in woodcuts accompanying the paper, for while that of the sound foot has a normal concavity on the inner side where the arch of the foot prevents contact with the ground, the whole sole of the right foot so touches the ground as to make one continuous line along its internal margin. The circumference of the right foot was found increased at the level of the cuneiform bones, and there was a well-marked projection of the dorsum, with a sensation of increase in the size of the first metatarsal and the internal cuneiform bones. The whole sole of the foot, however, seems somewhat shrunken. M. Féré enters into a consideration of the actual cause of the deformity, and looking into the construction of the arch of the foot points out that, from its form and build, destruction of the ligaments which retain the second and third cuneiform bones in place must lead to displacement upwards of one of them by the lateral pressure of the other two. He suggests that this displacement of a cuneiform bone is the real cause of the strange alteration in the shape of the foot.

The same journal (p. 486) also contains an account by Dr. Boyer of a case which fell under his care, and in which he had the good luck to be able to make post-mortem examination of the affected limb. The case is that of a man aged 45, in whom the symptoms began as long ago as 1870. A severe lumbago was then followed by violent lightning-pains, and in the course of a year or so by spontaneous dislocation of the right hip. After four or five years had elapsed, the man had extensive swelling of his right knee, which subsided and left the bones enlarged. About the same time both his feet were attacked in the same way, and were left enlarged and much deformed.

Not until 1879, however, did any ataxy become developed, but once begun, it rapidly increased. The patient died from "tuberculosis" in August 1883. Passing over other conditions, there were remarkable osseous changes in the right hip, in both knees and in the feet. The hip was completely dislocated, and the capsule of the joint was found converted into a thick osteo-cartilaginous shell, and there were numerous bony deposits—one especially large being just below the small trochanter, at the upper end of the femur, which with the ossified capsule formed a sort of new joint, the head and great trochanter having completely disappeared. The epiphyses of the tibiæ and femora had obviously undergone considerable atrophy, the spongy tissue thereof being soft, friable and vacuolated. The lesion of the feet was symmetrical, and consisted of enlargement of the astragalus, calcaneum and scaphoid, without anchylosis, and more notably of enlargement with anchylosis of the cuboid and three cuneiform, with their articulating metatarsal bones. The spongy tissue of all these bones was decidedly softened and friable. Yet another case of a like nature is to be found recorded in the same journal for May (p. 418), by MM. Gaucher and Duflocq, of "arthropathic tibio-péronéo-tarsienne gauche tabétique." At the age of 25 a soldier had a severe attack of cholera, and from that time onwards his disposition completely changed, and he became subject to attacks of great nervous irritability, and shortly afterwards, in 1850, to lightning-pains in the legs. About the same time, also, ataxy began, and, along with the pains, this continued in varying degree until 1869. In this year these symptoms became more prominent, and to them were now added anæsthesia of the soles, occasional diplopia, and loss of acuity of vision. During the siege of Paris in 1870-71 he rapidly got worse, became subject to hysteriform attacks, and occasionally had œdema of the lower part of the legs. During one of these complex seizures in 1881, the whole left leg became œdematous, but the swelling subsided after a few days' rest from all parts except the ankle and foot. Under prolonged treatment this indolent swelling of the soft parts almost entirely disappeared, leaving, however, very obvious bony enlargement of the lower part of the tibia. Again in the same year he had another attack of severe pain, followed by a like œdema, by a steady increase of the other symptoms, with incontinence now of both urine and fæces, and ataxy of the upper limbs. Leaving aside a minute description of the many symptoms present, the lesion of the ankle is thus described when seen in 1883. Tarsal and metatarsal bones are unaltered, with the exception of some enlargement of the head of the first metatarsal. The anterior part of the internal malleolus is much enlarged, and presents a uniform smooth swelling, which gradually fades away into the shaft of the tibia, which thus appears to have a well-marked curve inwards. The most noteworthy change, however, is in the fibula, which at its lower fourth begins to be enlarged, and increases in size downwards, so as to present a large osteophytic tumour on its anterior part, which is separated from the external malleolus by a depression, which thus gives it the appearance of being a second external malleolus. These cases all present certain common features. In all, lightning-pains were prominent; in none was ataxy a pronounced early symptom, nor were there true gastric crises. In each the bony changes were preceded by severe attacks of pain, associated with sudden œdema of the parts, and these changes seem in all of them to have consisted of atrophy here, and of the growth of exostoses there. In all, the changes themselves and manipulation of the affected joints were almost painless. The cases are a valuable addition to those which have been already published, and they go to show that the small joints and bones of the foot and ankle, though less often attacked, are liable to the same sort of arthropathic affections in the course of tabes dorsalis as are the larger joints of the body. The whole series of cases, here given in briefest abstract, forms an important contribution to the study of this marvellous disease, which seems daily to widen the domain of its symptoms, and which may come under observation when wholly unexpected, and in the most deceptive guise. It is not without interest to note, that in four of the nine cases recorded in these various papers there is a clear history of syphilis, and that in none of them is any special mention made of rheumatism, either in the patients themselves or in their families. To the existence of this latter disease in those afflicted with arthropathies it would be well if future observers were to direct very careful attention, for the special character of these so-called Charcot's joint diseases is, as it is well known, doubted by very competent observers, and this point not less than others has yet to be cleared up with reference to the ætiology and pathology of tabes dorsalis.

Dubler on Neuritis in Herpes Zoster.—In an abstract of a previous contribution of MM. Pitres and Vaillard on "Non-traumatic Peripheral Neuritis" (see 'BRAIN,' Vol. VI. p. 555), reference was made to a case of theirs, where inflammatory degeneration of two intercostal nerves was regarded as the cause of attacks of Herpes, and to their belief that microscopical examination had been too exclusively directed hitherto to central structures rather than to the nerve-trunks themselves.

Dr. A. Dubler now brings forward (Virchow's 'Archiv,' May 1884, p. 185) two cases of great interest, where by very careful examination the presence of peripheral neuritis was as clearly established as was the absence of central disease. In one, the inflammation of the intercostal nerves was clearly due to their being involved in some periosteal swellings of the ribs, from which points of irritation the degenerative changes had spread and involved their very finest endings both in muscle and skin. The cause of the neuritis was not so obvious in the other case, but the peripheral changes were not less pronounced. In this second case it is noteworthy that the spinal ganglion of one of the affected intercostals—the 9th—showed degenerative changes, but only in that part of it to which the degenerated fibres of the trunk itself could be traced; and as no such changes were to be found in the ganglia of the other diseased nerves, it seems probable that the neuritis may have begun as spontaneously in the ganglion as in the trunk, and that there was really no connection between the two. Plates are given of the microscopical appearances, which are seen to be of the same nature as have been described by Pitres and Vaillard. M. Dubler's paper is worthy of careful perusal, for in addition to his own cases, recorded at great length, he refers to numerous others which have been published, and he shows how really inadequate were the examinations made in them, and how necessary it is to subject the nerve-trunks themselves, throughout their whole lengths, to microscopy. It is indeed somewhat remarkable that the discovery of these peripheral changes is of comparatively recent date, for the suspicion has long existed that some change in the nerve-trunks themselves must underlie that form of Herpes which accurately follows the line of nerve-distribution. As long ago as 1869, Broadbent wrote in a paper on "Speculations concerning Herpes Zoster," that the "provisional hypothesis" at which he had arrived as the proximate cause of Herpes Zoster, was that there was "some undefined condition of a nerve-trunk, whether inflammatory or anæmic or other he was not prepared to say, or how it was induced, and that the particular part of the nerve affected was certainly not the root."

Schulz on the Importance of Tendon-reflex in the Examination of Suspected Simulation of Spinal-cord Diseases, (*Deutsches Arch. für Klin. Med.* Feb. 1883, p. 455.)—After giving a careful *résumé* of the different views which are held on the physiological import of the so-called tendon-reflexes, Schulz comes to the conclusion, from the clinical observation of numerous cases, that simulation of ankle-clonus or of the absence of patellar tendon reflex (knee-jerk) is a thing absolutely impossible. Two striking objective phenomena can thus, regardless of the patient's will, be always investigated in cases of suspected simulation of paralysis; and Schulz points out the value of this observation in cases of alleged injury from railway accidents. The instances have not been few in this country where the presence or absence of these phenomena has afforded invaluable aid in diagnosis when no objective sign could be discovered amongst overwhelming numbers of subjective symptoms, and Schulz tells how in Germany also it is especially after railway collisions that simulation of paralytic maladies is most likely to be found. Railway and post-office servants have learnt, he says, from one another the complex symptoms of the *railway spine*, and it is therefore most important for the surgeon to be armed with some means of diagnosis which is practically conclusive. Hardly sufficient stress is laid, perhaps, on the fact that both ankle-clonus and exaggerated knee-jerk may be sometimes met with in paralytic conditions not dependent on organic disease of the spinal cord or in certain functional states of the nervous system, but there is every ground for believing that true ankle-clonus and abolition of knee-jerk are absolutely beyond the active will of the patient. There yet remains a small percentage of cases, so small as scarcely to lessen the value of the phenomenon—where the knee-jerk is normally absent. Berger, for example, found it absent in 22 out of 1409 presumably healthy men, and in 694 school-children of both sexes examined by Block it was wanting in 5, of whom 4 certainly belonged to neurotic families, one of them, moreover, having curvature of the spine, and only one being absolutely sound. The difficulty of eliciting the knee-jerk is, moreover, great in short thick legs, as has been pointed out by Westphal; and perhaps we are as yet ignorant of some possible variations of the so-called reflexes within the limits of perfect health.

Herbert W. Page.

Remak on Hypoglossus Spasms. (*Berl. Klin. Wochenschr.*

1883, No. 34.)—A man, 33 years old, suddenly felt numbness in the left half of the tip of the tongue, taste, feeling and movement being unaffected at first; the numbness spread in two weeks to the middle of tongue, the inner side of left half of lower lip and corresponding gum, and at the same time he had painless spasms of tongue, affecting him in speaking and eating. Slight paralysis of left facial nerve, as shown by lowering of left side of mouth, was present; the spasm of the tongue consisted in a flattening and narrowing of the upper surface, and rapid protrusion, forty times a minute, while corresponding rhythmical contractions of the muscles of tongue between the lower jaw and the hyoid bone occurred, and affecting especially the genio-hyoid muscles. Movements were increased when the tongue was protruded. Otherwise voluntary movements free in all directions. Sensibility normal; teeth healthy. Taste and sensibility of all kinds normal. Electric excitability normal; by tetanizing left side of tongue the spasms ceased. Speech not altered, except thick and slow, and talking did not increase the movements. Has often bitten his tongue. Movements increased by eating and not by drinking. Velum palati normal.

After some days he had severe clonic and tonic convulsions of the left side of the face, especially the buccinator, zygomatici, triangularis and quadratus menti, digastricus and platysma myoides muscles, while the upper branches of facial nerve were not affected; the patient was prevented from speaking or eating for one minute while this attack lasted. After these attacks the movements of the tongue were lessened, but the paresis of left side of face was rather more. Subsequently several convulsions occurred five or six times a day on eating and also spontaneously, while the original movements of the tongue stopped altogether. Remak quotes Romberg's opinion, that hypoglossus spasm associated with mastication is the rarest form of the disease.

He refers to cases of hypoglossus spasm produced by attempts to articulate. Also to one of Weir Mitchell's, with painful spasm of the tongue, spreading to the facial nerve, and cured by the extraction of a tooth.

From absence of pain or any peripheral irritation, a reflex cause cannot be substantiated. He considers that the lesion might be in the hypoglossus nucleus, and also in the left facial nucleus contiguous to it; or in the right ascending frontal convolution of brain, although there is no aphasia and no loss of sensation. Treatment such as by galvanic current and nerve-stretching would be influenced by a correct regional diagnosis and by determining whether the disease is functional or organic.

The attacks diminished, and were finally cured in eight days by treatment with iodide of potassium, bromide of potassium, and daily stabile galvanic treatment with the anode pole to the infra-maxillary region and left side of neck and throat.

Rumpf on Influence of Narcotics on the tactile Sensibility of the Skin. (*Vortrag 2ter Med. Congr. Wiesbaden,* April 1883.)—He employed hypodermic injections of hydrochlorate of morphia, from ¼ to ⅓ of a grain, and in 6 to 7 minutes a diminished cutaneous sensibility over the whole body was noticed which reached its maximum in an hour; the sensibility was tested by an æsthesiometer. He agrees with Jolly—and so differs from Eulenburg—that sensibility is not more diminished at the point of injection, than over the rest of the body; but he thinks that under pathological conditions the local effect might be stronger. Chloral hydrate, given by the mouth, produced less effect than was expected, and after a dose of 30 grains, sufficient to produce drowsiness, only a very slight diminution of sensibility was obtained. Alcohol by the mouth in doses of about 1½ ounces diminished the sensibility, particularly in the lower limbs. Bromide of potassium in drachm-doses gave the most decided diminution of sensibility. Extract of hyoscyamus had, contrary to expectation, no particular effect. Cannabinum tannicum, the glucoside of Indian hemp, in doses of 4 grains by the mouth, caused considerable lessening of sensibility, and also produced calm sleep. Caffein, ⁴⁄₈ to 1¾ grain in an ounce of coffee had a very strong influence in increasing the sensibility of the skin, but the effect soon passed off. Morphia had the most effect, and bromide of potassium next.

Kast on Ataxic Movements in disease of the Spinal-Cord (*Nat. Forsch. Gesells. Freiburg*).—He discusses the theory of co-ordination, mentioning that there exists a hypothetical system, which receives motor impulses from the cortical centres, and directs them to the various groups of muscles; and if these motor impulses are deficient, owing to a lesion of the co-ordinating systems, or to some hindrance in the peripheral tracts, faulty combination is the result, and hence ataxia. He asks whether correct co-ordination is due to centripetal sensory impressions, or to purely motor impulses. Ataxia occurs in tabes, Friedreich's disease, disseminated sclerosis, and ataxia.

He quotes cases of Kahler and Pick, and Vulpian, with multiple tubercles of cord in the case of former, and spinal compression in that of the latter author, associated with ataxic movements. He gives in extenso the case of a man who had fracture of the dorsal vertebræ from a fall, extravasation in the dura mater, myelitis of the dorsal cord from compression, complete motor paraplegia, slight loss of sensation, gradual motor recovery in eight weeks, but with typical ataxia of legs, ending in complete recovery. When the ataxia was developed he had full motor power in the legs. In the right leg ataxia and loss of sensibility to touch and pain were more marked than in the left. Ataxia was not much increased on closing the eyes. Both knee-jerks readily obtained; no ankle clonus; plantar reflexes active; sphincters not affected. Left leg recovered before the right.

In another case of acute transverse myelitis of the dorsal cord, with complete motor and almost complete sensory paralysis of both legs to touch, temperature and pain, incontinence of urine and fæces, there was decided ataxia with return of motor power. Complete recovery in eighteen months. In this case the plantar reflex was active, cremasteric absent; knee-jerk well obtained, left more than right; ankle clonus on left, not on right. When he had a complete return of motor power the ataxia persisted, and was more marked in the right than the left leg.

Kast remarks that, in these two cases, a stage intervened between the complete motor paralysis and return of muscular power, in which, with almost complete motor power, there were considerable defects of co-ordination and ataxia. In these cases he thinks that either the extensive myelitic process involving the whole transverse section of the cord, had disappeared from all parts, leaving its effect most in the posterior columns containing the co-ordinating tracts; or that here the lesion was from the beginning most intense; or that for some reason, lying outside this tract, its recovery or that of its collateral fibres had been unduly retarded. He draws attention to the laxed condition of the muscles and joints, which was very marked in this patient, and which has been considered an explanation of the ataxia in these cases.

He quotes Tschirjew, who considers that the tracts for the preservation of muscle tonus are identical with those for the conduction of tendon reflexes; and the failing of the muscle tonus of the antagonizing muscles, produced by the interruption of the centripetal nerve tracts, causes the excessive and irregular movement of the limbs; and this disturbance of movement produced by the lesion of these peripheral regulating apparatus, he calls peripheral ataxia. He also thinks that the absence of the tendon reflexes is caused by a slighter lesion of nerve fibres, than are the loss of muscle tone, and the ataxic movements of co-ordination, due to the tonus of antagonizing muscles; and thus tries to explain the incongruity of the presence of the tendon reflexes with the disturbance of co-ordination. Westphal and Senator oppose this theory; and Kast thinks that his cases prove, that absence of tendon reflexes is not necessary for ataxia.

Kast on Reflex Saltatory Spasm. (*Neurol. Centrbl.* 1883, No. 14.)—Patient, a man 39 years old, with no neurotic family history, complained of convulsive "drawing" of both legs from the calf upwards. Onset sudden. One morning he could not stand still on his feet, as soon as they touched the ground the heels lifted up and caused him to stumble and fali. There was some increase of patellar reflex; he got better after being in bed a month, and resumed work. Subsequently, when very tired from walking, he suddenly had cramp in the calves, severe shaking of both legs, and fell down, and the shaking occurred whenever he tried to walk. After seven weeks in hospital, with warm baths and constant current, he got better, but the same symptoms came on again when he was tired. When put on to his legs, the muscles contract rhythmically and his heels are raised off the ground, the shaking extends from the calf to thighs and buttocks; he staggers and falls. Muscular power intact. No tremor on voluntary movement when lying down, no inco-ordination, no rigidity. Sensibility normal. Muscle sense and electric state normal.

Patellar tendon reflexes excessive on both sides. Ankle clonus present, but sometimes prevented by a slight tetanic rigidity of the calf-muscles. Skin reflexes increased, plantar abdominal and cremasteric, in both lower limbs increased *tendon reflexes.* He got better with a generous diet, and was able to walk in ten days; but clonus of the calf-muscles could be produced when he was made to stand on his toes; tendon and skin reflexes gradually diminished till ankle clonus disappeared. Kast states that only nine cases have been noticed. He quotes Erb in support of the theory, that this disease is probably due to increased reflex irritability and especially of the tendon reflexes, and is rather a symptom of different diseases than a distinct form of spasm. Refers to a case of Petrina's, where the patellar tendon reflex was very weak, and later on was not obtained, and ankle clonus was absent throughout; but this case was probably an acute disseminated cerebro-spinal disease. In Kast's case the spasms could sometimes be stopped by compressing the muscles or by cutaneous electric irritation, but not by voluntary impulses.

The term of reflex saltatory spasm was first given to these cases by Bamberger. Kast thinks that the saltatory spasm can occur as part of an acute disturbance of co-ordination, and can exist when there is extensive diminution or even absence of the skin and, tendon reflexes.

C. E. Beevor, M D.

Associated Systematic Sclerosis of the Columns of the Cord.—In the *Archives of Neurology* (January, 1884) Drs. Ballet and Marie report a case of sclerosis of combined systems of the cord which presented clinical and pathological aspects of much interest. The patient, four years prior to admission into Salpêtrière, suffered from amblyopia, and became completely amaurotic within a year, with the accompaniment of severe headaches, compared to the pressure of an iron band around his head: there was no vomiting. Three years subsequent to this, "lightning pains" began in the limbs, and cutaneous hyperæsthesia of the extremities rendered the least touch painful. Close questioning, however, elicits the fact that such pains might have occurred previous to the development of ocular symptoms. At this period (1881) the arm became first enfeebled, and three months later there was loss of muscular power in the leg.

Upon admission in February, 1882, he was bedridden from paralysis of the limbs; he was also completely blind, not distinguishing light from darkness. The ophthalmoscope revealed grey atrophy of the papilla. He complained of transient buzzing noise in the ears, with vertigo.

Motility.—There is advanced paralysis of upper and lower limbs, cannot walk or even stand erect; executes feeble movements, but with much effort while recumbent. Arms are slightly less affected than the legs. The paralysed limbs are rigidly contracted and the seat of cramps: deep reflexes notably exaggerated: muscles wasted: no paralysis of bladder or bowel.

Sensibility.—Transient, often recurring pains of burning character affect the legs, and are accompanied by numbness of feet; all the limbs reveal painful hyperæsthesia of the skin, except left leg, which is partially anæsthetic. Later on in the year, lancinating pains affect the chest, and are often localised in one spot in an intercostal space. Frequent syncopal attacks occurred during this year; and there is now loss of power over sphincters of bladder and bowel. For five days preceding his death he suffered from sharp pyrexia, ranging betwixt morning and evening from 102° to 103° Fah.: an eschar forms on sacrum: skin becomes citron-yellow, and the conjunctivæ of icteric tinge.

Autopsy.—Brain shows no disease beyond grey atrophy of optic nerves, chiasma and tracts, with greyish degeneration of the outer geniculate bodies.

Spinal cord, upon microscopic examination, shows a sclerosis involving both posterior and lateral columns: in the greater part of the lumbar cord these columns have escaped, but higher up nearly their whole thickness is invaded. It is only in the lowest cervical and highest dorsal region that the columns of Goll are completely altered, and the whole thickness of the posterior columns affected by disease: higher still, the columns of Burdach are untouched, save close to the pia mater; but the columns of Goll are utterly degenerated as far as the medulla oblongata.

There is sclerosic change of the lateral columns at all heights as far as the pyramidal decussation; forming a small wedge-shaped tract in the lower lumbar, it rapidly enlarges in the upper lumbar region, and from this point up to the decussation it involves nearly the whole depth of lateral columns, including the direct cerebellar and crossed pyramidal tracts. No change had occurred in the cells of the cornua.

Under strong objectives the following features are revealed in the diseased tracts:—

(*a*) Fresh preparations show an absence of granular and fatty bodies. (*b*) Sclerosis is not of the same degree at different sites: *it is denser next the pia*, and less dense in the posterior than the lateral columns. (*c*) Amyloid bodies are found at margin of diseased tracts. (*d*) Proliferation of neuroglia nuclei. (*e*) Numerous stellate cells, especially near peripheral parts in the lateral columns. (*f*) Large size and number of vessels, with walls and sheaths infiltrated with leucocytes. (*g*) Where the nerve-tissue is not completely destroyed, the morbid change resembles that of acute and subacute forms of myelitis, viz. "distended nerve-sheaths and varicose axes." (*h*) Anterior and posterior nerve-roots were normal. The optic nerve showed complete absence of nerve-fibres.

Our authors, upon reviewing these data, argue that this case is not one of systematic myelitis, in the usual acceptation of that term. They emphasise the strange predilection shown by the morbid process for the dorsal region of the cord, and the integrity of radicular zones at lower lumbar realms — for tabes by preference attacks the lumbar cord. Then, as regards the lateral columns, they urge that authors frequently use as synonymous the terms "lateral" and "pyramidal" columns, instead of clearly distinguishing betwixt the centrifugal and centripetal tracts of this complex system. They insist upon the advance of sclerosis from the meningeal border of the cord inwards, through the cerebellar to the crossed pyramidal systems—breaking through physiological barriers and diffusing itself irregularly with an independence of behaviour which recalls the progress of multilocular sclerosis: it is *not*, therefore, a systematic but a diffuse sclerosis.

It is difficult to conceive a genuine systematic sclerosis of the lateral columns which should absolutely respect the integrity of the columns of Turck and anterior horns: yet M. Jubineau records three observations of such a character. It is of interest to note the atrophy of optic nerves which so frequently accompanies ordinary tabes, associated with a lesion of the posterior columns, which in this case appears developed by a process wholly differing from that of ordinary locomotor ataxy. The authors then summarise what appears to them to be various types of system-affections due to sclerosis of the posterior and lateral columns of the cord: these are as follows:—

1. Diffuse sclerosis, as illustrated by the case under consideration.
2. Classical ascending sclerosis, involving the columns of Burdach, Goll, and direct cerebellar tract, in virtue of their conductibility and physiological functions.
3. Combined case of primary systematic sclerosis, with a consecutive diffuse myelitis.
4. Systematic sclerosis, consecutive to diffuse interstitial myelitis.
5. Combined ascending and descending systematic sclerosis.

The widely dilated vessels surrounded by leucocytes is insisted upon as suggestive of a perivascular origin for the changes seen: as in the liver, so in the cord, the forms of sclerosis must be various. They suggest "peritubular" and "perivascular" as appropriate terms, since the terms specifying the sclerosis should indicate its point of departure; of course every perivascular sclerosis eventually involves the medullated tubuli and becomes peritubular. In connection with this statement, it may be remarked that a case of tabes in conjunction with ophthalmoplegia externa was published by Dr. Buzzard in the April number of 'BRAIN' for 1882, in which similar notable lesions of the blood-vessels of the cord were described, and a second case by him in the January number of 'BRAIN' for last year, revealed such changes as induced him to regard the affection as primarily a peri-arteritis in origin. Prof. Adamkiewicz is regarded by Drs. Ballet and Marie as in error when he refers the classic form of locomotor ataxia to lesions of a perivascular connective change, rather than a periradicular implication, and they incline to support Charcot's and Pierret's views upon this point. In the case under consideration, a specific origin is claimed, there being a history of syphilis, and all similar cases are referred to probably some intravascular toxic or infective agency.

The authors next refer to the antagonistic nature of symptoms which may be supposed to arise in the combined systematic forms, viz.:—the acute pains, hyperæsthesia or anæsthesia of a posterior sclerosis, with its abolished reflexes and flaccidity of limbs; and the rigid contractures and exaggerated reflexes of a lateral sclerosis. In a case quoted by M. Babesiu, there were at first feebleness and heaviness of the legs: contractures, followed by pains in limbs and genitals: next, atrophy of disc towards the end, plantar anæsthesia, loss of muscular sense, paralysis of bladder, tendon-reflexes slightly exaggerated. In a case given by M. Raymond, the predominant symptoms were those of lateral sclerosis, although both lateral and posterior columns were affected. In the three cases, therefore, of Raymond, Babesiu, and that by Ballet and Marie, the lateral column maintained its rights to assert its implication by contractures and exalted deep reflexes, despite the grave lesions in the posterior columns. We note here, however, that in Babesiu's case the tendon-reflexes were but "*slightly exalted*," and it appears more natural to suppose the various degrees of modified reflexes would occur in these cases of combined systematic sclerosis sometimes more, sometimes less exaggerated, or more or less diminished, according to the predominance of the one or the other lesion. Dr. Buzzard, in his 'Lectures on Diseases of the Nervous System,' has specially drawn attention to the question propounded by Ballet and Marie. Our author's explanation of the phenomena is at all events ingenious: the columns of Burdach are supposed to be free from lesion in the upper cervical and greater part of lumbar regions; hence the reflex arcs are free for both extremities. The explanation is therefore one of *localisation.* So for lesions predominating in the posterior columns, the advantage being with the degree of lesion, so to speak, if the whole height of the cord be affected there will be abolition of reflexes, and the limbs flabby or but slightly contracted; if, on the contrary, a large part of the posterior columns be intact, the sclerosed lateral columns maintain their rights to their usual mode of symptomatic expression. This is nearly Westphal's opinion: "When there exists an associated affection of the posterior and lateral columns, neither muscular rigidity nor contraction supervene if the lesion of the posterior columns extends as far as the lumbar enlargement, and if the radicular zone of these parts be degenerated."

Hemiplegia occurring during the course of Diabetes. (Drs. Raymond and Artaud: *L'Encéphale*, No. 3, 1883.)—It is generally admitted, since the researches of Claude Bernard, that certain lesions of the cerebro-spinal axis may induce glycosuria, whilst the latter affection in its turn may react on the nervous system, producing very varied symptoms of centric disturbance. Equally well is it established that amongst the most important accompaniments of diabetes are the initial monoplegias characterised by their transient, mobile nature, their incompleteness and tendency to affect individual musculatures, and the later hemiplegic seizures of more persistent type, often preceded by intellectual troubles like those of cerebral softening, and usually due to vascular lesion. The patient whose case is detailed was a female aged 68, with nothing peculiar in her former history until three years prior to admission, when very intense irritation of the genitals, with frequent copious micturition, commenced, accompanied by intense thirst.

Twelve months later, dimness of vision affected first the right and then the left eye. Upon admission, the lenses of both eyes were cataractous and patient nearly blind; there was no longer intense thirst; appetite and digestion were normal; there was obstinate constipation. Physical examination reveals very little wrong with chest or abdomen, but the urine is found to contain on an average 15 grammes of sugar per litre, and heat and nitric acid show much albumen. For a period of nine months this condition is maintained, but also supplemented by severe lancinating pains at the right occiput and progressive mental enfeeblement. The daily amount of urine passed, from 1500 to 2000 grammes, contained besides the usual amount of sugar about 1½ grammes of albumen per litre. The left arm is now affected by a progressive paralysis, which slowly invades the leg also on this side; there is no facial or ocular paralysis.

The left arm is not completely paralysed; the grasp is, however, very feeble; sensibility to pain is somewhat diminished; reflexes and electric-contractility are normal. The condition of the leg is similar, except that the deep reflexes were exaggerated. The autopsy revealed a focus of softening in the right hemisphere, limited in front by the ascending parietal, below by the first temporo-sphenoidal, above by the postero-parietal and behind by the annectent gyri; the membranes adhered to the diseased part; no other portion of the hemispheres, pons and medulla or cord, presented any coarse change. Microscopic examination of the bulb and cord shows none of the changes described by Dickinson, and no secondary degenerations of crossed or direct pyramidal tracts. The organs, however, generally (liver, heart, kidneys) presented a notable degree of sclerosis spreading along the perivascular tracts. Strangely enough, the cut illustrating the article exhibits the lesion upon the left hemisphere.

Raynaud on a case of Cerebral Localisation.—Dr. Raynaud in the same number of *L'Encéphale* details a case bearing upon cerebral localisation. The patient, when admitted, suffered from right hemiplegia with incomplete aphasia (replies usually by gestures, sometimes by inarticulate sounds, interspersed with distinctly uttered words, such as "non," "coquin," &c.).

Three weeks later, a second apoplectic seizure with deep coma occurs, from which she recovers gradually, retaining pantomime and the former expressional power; but a few months later on she is found to have *entirely lost* the faculty of articulate language, and a subsequent apoplectic seizure with convulsions terminates fatally. The autopsy reveals,

1. An old superficial hæmorrhagic focus, which has involved the posterior part of the third frontal, the junction of central gyri below, and the whole of the insula.

2. A recent hæmatoma of the dura mater, spreading over the posterior frontal, the parietal and the temporo-sphenoidal gyri, enclosed in a false membrane.

3. A softened disintegrated condition of the external capsule and caudate nucleus, spreading inwards to these parts from the site of old hæmorrhage over insula.

To the first of these lesions M. Raynaud attributes the hemiplegia with partial aphasia; to the second, the subsequent apoplectic seizure; whilst to the extension of softening into the caudate he attributes the complete loss of articulate language which supervened when these, the evidently latest lesions, occurred.

Magnan on certain peculiar features in Bilateral Hallucinations. (*Archives de Neurologie*, November 1883.)—A more unusual form of hallucinatory manifestation than the purely unilateral kind described by Calmeil, Moreau, Michéa, and others is a bilateral hallucination, in which the character or colouring of the subjective image varies with the active hemispheres: with the right ear, for instance, the patient fancies he hears pleasurable and encouraging sounds and words; with the left, he only hears abusive epithets, threats or doleful sounds. A study of such cases much strengthens the view of the dual nature and functional independence of the cerebral hemispheres. He quotes the case of a blacksmith, aged 34, of strong neurotic inheritance through epileptic and alcoholic parents, who developed epilepsy at the age of fourteen: preceding his convulsive seizure he always experiences "a taste of blood in the mouth": after the attack he is torpid, or partially unconscious; he performs various automatic movements, or becoming excited, violently assaults, and on one occasion nearly strangled a woman near him. In childhood he had been the house-drudge, and was neglected; later in life, his epilepsy being a barrier to marriage, he became irritable, sensitive to slights, and developed ideas of persecution: at the age of twenty-six he began to suffer from auditory hallucinations affecting the *right side*; he heard abusive epithets uttered, and imagined the devil conversed with him. Gradually, however, ideas of a different order developed: he believed he was to possess a great fortune, which had been accumulated by seven generations: at night he would gaze at the sky, and question the moon and stars; and becoming more and more arrogant, accounted himself to be the Son of God, and would indulge in varied apothegms, e.g., "The smallest is the greatest:" "The lowest is the highest:" "Poverty is Wealth." The abuse lavished upon him previously now lessened, and words indicating praise and eulogy were heard by him, but this upon the *left side*: he often heard the sound of ringing, which, if heard upon the right side, would be interpreted as of bad omen; if heard upon the left side, would cause pleasurable anticipation. For two years gay or ambitious hallucinations predominate, and such conceptions always prevail upon the left side, or, in other words, are referred to that side for their assumed objective explanation.

Another patient, aged 48, of intemperate habits, who had frequent attacks of delirium tremens accompanied by hallucinations of a sad, sombre colouring, believed himself insulted, saw cats, rats, birds, &c., about him, and was annoyed by bad odours. As the delirium in this case subsided, and he became calm, he for a long time retained delusions of persecution, and heard a voice constantly using insulting terms on the left, whilst with the right ear he fancied he heard a voice using consolatory expressions.

A patient, aged 34, of neurotic diathesis, father alcoholic, brothers alcoholic, three sisters hysteric, he himself a heavy drinker, had two attacks of delirium tremens. He suffered long after subsidence of delirium from hypochondriasis and fears of poisoning: the right side appeared to receive instructive remarks upon God and the Virgin; the left side was the source of ribald and obscene language.

In the fourth case, aged 35, the son of a drunken and suicidal father and hysteric mother—who himself, of a sad, morose temperament, contracted intemperate habits at an early age—was addicted after drinking bouts to desperate suicidal attempts by every possible means. During the last five years he had had several sharp attacks of alcoholic delirium, lasting two or three days, and accompanied by the usual hallucinations. In the intervals he retained fears of ill-treatment, hearing abusing epithets on the left side, whilst encouraging words and sentiments of approbation were heard from the right side. In these four cases cited, no peripheral lesions of the ears were detected, and auditory acuity was perfect and equal on both sides. After citing a remarkable case, where experiments were tried upon a hysteric patient hypnotised, in which this dual control of the cerebral hemispheres was confirmed, Magnan discusses the site of origin of such hallucinations, and he agrees with Tamburini that they depend for their origination upon an excitation of the cortical sensorial centres.

He summarises his views under these heads :—

a. Bilateral hallucinations, varying in character according to the side affected, are not dependent upon peripheral lesions of the auditory apparatus.

b. They do not differ from ordinary hallucinations in their onset, evolution or general characters.

c. They constitute a new argument in favour of the double nature and functional independence of the cerebral hemispheres, and they have for their site of origin the cortical sensorial centres.

d. Experiments during the hypnotism of hysterics confirm in all respects the results of clinical experience.

W. Bevan Lewis.

Nutritive Alterations and Deformity of Fingers from Pressure on Nerves in the Axilla.

By F. T. Miles, M.D. (*Journal of Nervous and Mental Disease*, Oct. 1883).—The patient, a female, sustained a fall when walking, two years before the present observations were made, and her left humerus was dislocated downwards into the axilla. When discovered, the dislocation was irreducible. She had much pain in the arm, shoulders and fingers, tenderness about the shoulder-joint and above the clavicle. There was numbness of the ring and little fingers; the little finger was deformed and its joints stiff, the nail distorted and crumpled up; the skin of the little and ring-fingers was pale, glossy, and looking as if stretched. The nails of the other fingers were curved, both laterally and longitudinally. Sensation was not lost, but blunted. Farado-contractility of the muscles was normal in the forearm; slight in the interossei, absent in the muscles of the little finger. The author thinks, that the case suggests that trophic nerves exist as apart from motor or sensory nerves, and that they are the more vulnerable under certain circumstances.

The Visual Area in the Brain determined by a study of Hemianopsia.

By M. Allen Starr, M.D. (*American Journal of Medical Sciences*, Jan. 1884).—The author, by a series of 32 cases, derived from various sources, seeks to establish the conclusions of Munk, that the occipital lobes are necessary for the perception of visual impressions, and that each occipital lobe is related to both eyes in such a way, that corresponding halves of both retinal areas are projected upon the cortex of the occipital lobe of the like named side, e.g. destruction of the left lobe produces loss of function of the left halves of both retinæ. The course of the optic fibres is, he maintains, from the commissure (where a partial decussation has taken place) backwards to the corpora-geniculata, pulvinar, and corpora quadrigemina, and thence without further decussation by a tract passing backwards from the thalamus to the occipital lobe. This is in opposition to the scheme formerly proposed by Charcot, which supposes that a second semidecussation takes place beyond the corpora quadrigemina, so that all the fibres from one retina reunite in the cerebral cortex of the opposite side; and it is in opposition again to the still more complicated scheme of Grasset.

Ferrier's theory, that the cortical centre for vision lies in the angular gyrus, is not sufficiently supported (the author thinks) by post-mortem evidence. He suggests that, as the tract of fibres from the thalamus to the occipital lobes comes near the surface in the neighbourhood of the angular gyrus, some cases, apparently of lesion to the angular gyrus only, may have really involved also this tract of fibres.

The author's cases are grouped in two series: (α) 5 cases of hemianopsia, with lesion of one optic thalamus; (β), 27 cases of hemianopsia, with lesion of the occipital lobe. There was in some of these more than one lesion. He draws the following conclusions from them: Lateral homonymous hemianopsia may be produced not only by a lesion of one optic tract, but also by a lesion situated either in the pulvinar of one optic thalamus, in the posterior part of one internal capsule, or its radiation backward towards the occipital lobe; in the medullary portion, or the cortex of one occipital lobe. Since in the majority of cases considered, the hemianopsia was limited by a vertical line passing through the fixation point, it is impossible to say from the hemianopsia only at which part of the course of the optic fibres the lesion lies. As regards associated symptoms in these cases; hemiplegia was noted in 13 cases, hemianæsthesia in 8, aphasia in 10, monoplegia in 4; these could be accounted for by other cortical areas or other systems of fibres being affected; the author thinks that no symptoms except the visual are to be referred to the occipital lobes.

Cases of Union of Peripheral Nerve Trunks. By S. Weir Mitchell, M.D. (*American Journal of Medical Sciences*, July 1883).—*Case* 1.—Peculiar nutritive changes of palm and back of hand, ascribed to milking; herpes; neuralgia. Elongation of median nerve; section of radial.

A female, æt. 40, had had for ten years numbness in the right palm, first noticed while milking cows, later while ironing; afterwards pain in the palm, and in all the fingers of the right hand. During the last two years the pain prevented her using the hand. It was most marked in the morning on waking, made worse by cold, relieved by heat or pressure. Three months back, herpes appeared on the back of the hand; and for some years she had had bronzing of the face and of the breast, like that seen in Addison's disease. When examined, the pain was found to be situated in the palm, between the second and third metacarpal bones, extending through to the dorsum. There was slight loss of muscular power, no anæsthesia, no trophic disturbances beyond the herpes. Some relief was experienced from strychnine and lactate of iron.

Four months later, ulceration had set in on the site of the herpes: whenever this healed, the pain became severe. This was relieved by galvanism, but other ulcers appeared in various positions on the hand and forearm. There was defective sensation (after the attacks of pain), covering most of the skin in the median district, and some in the ulnar and radial.

The median was stretched. During the operation the pulse became slow, hard, and full. Two hours later the temperature of the hand rose. Four days later the areas of defective sensation had spread in the median ulnar and radial districts. The pain and ulceration, relieved at first, returned in a month's time.

The radial nerve was then divided. There was a similar effect on the pulse and the temperature of the hand. The area of defective sensation was extended further, chiefly up the wrist and forearm; sensation was quite abolished over one patch at the back of the hand. Nevertheless these variations did not wholly correspond to the radial distribution. The severe pain in the hand and the ulceration was completely relieved; some aching only being left in the hand, and in the neck and shoulder.

In *Case* 2 a neuralgia had existed for some years in the left supra-orbital nerve, and had for some years affected the infra-orbital branch, and at times the infra-maxillary. Section of the supra-orbital branch was ordered, but by a misunderstanding the infra-orbital was divided instead. The result, however, was to cure the pain in all the branches of the nerve.

In *Case* 3 resection of the left inferior maxillary had been performed for neuralgia, but the pain had returned, and the cutaneous sensation had been restored. Resection was again performed, and the canal plugged with dental cement.

Case 4.—Affection of nerves of left arm; remarkable lowering of temperature.

A man, age 51, letter-sorter, had been obliged, in consequence of an injury to his right arm, to make a disproportionate use of his left hand. For a year he had noticed a certain loss of power in the fourth and fifth fingers, and subsequently the same thing in the other fingers, with pain in the forearm: For six months, pain from the shoulder to the finger-tips on the approach of storms. Sensation to touch and to pain defective in the hand and lower forearm. All the hand-muscles somewhat wasted; power of extension in third and fourth fingers imperfect, excitability to galvanism and faradism diminished, but without qualitative changes; tendon-reflexes increased; skin smooth (not absolutely glossy), mottled red or uniform dull red. Temperature of the affected hand was lower than that of the other, once as much as 14° lower; the axillary temperature remained normal on the affected side.

Case 5.—Dislocation of left humerus into axilla, immediate and increasing nerve lesions; unusual nutritive lesions resembling abscess; extravasation of blood.

A woman, aged 24, dislocated her left humerus into the axilla; severe pain, with tingling to the finger-ends, set in at once, the fingers became weak the next day. The dislocation was reduced on the sixth day, but the pain and paresis increased. In a month the back of the hand and arm, chiefly on the ulnar side, began to swell, and then the palm, and finally blood was extravasated. In four or five months this swelling had disappeared, and sensation was normal except in the palm and in the palmar surface of the fourth and fifth fingers, where the skin was shiny red and hypersensitive; the muscles of the ulnar district were wasted. The nails were notched; the joints of the fingers, the wrist, elbow, and shoulder, were affected. The changes in electro-excitability appear not to have been limited to the muscles supplied by the ulnar nerve.

Multiple Cutaneous Ulceration. By J. EDMUND ATKINSON, M.D. (*American Journal of Medical Sciences*, Jan. 1884).—A half-breed child, aged 28 months, was attacked with itching all over the body and swelling of hands and feet. Next there formed, on the toes, fingers and hands, pimples which became vesicular and then suppurated, forming finally deep irregular ulcers. Diarrhœa and wasting set in, and ulceration of the mouth and tongue; one ulcer upon the face reached from the mouth nearly to the eye. It seemed probable, from the way in which the child scratched and bit the raw surfaces, that the affected parts were anæsthetic. Next, the forefinger of the right hand began to ulcerate deeply and rapidly, the parts seemed to melt away, the whole bone being bared in three days. Under careful dieting and tonic treatment the child recovered. No constitutional taint, such as syphilis or scurvy, could be found to account for the symptoms. The child had been well fed and cared for. It was not under the influence of mercury nor of ergot. The author quotes three somewhat similar cases. He believes the symptoms to be caused by some change in the central nervous system, either of the vasomotor or trophic system of nerves.

Case of Brachial Monospasm and Monoplegia with Sarcoma of Ascending Frontal Convolution. By W. R. BIRDSALL, M.D. (*Archives of Medicine*, June, 1883).—A woman aged 44, had had a growth removed from the thigh a year previously; this had returned, with glandular enlargement. During the last two or three months headaches, then vomiting and vertigo; finally fits commencing with jerking of the left arm (and left cheek?) and followed by paralysis of the left arm. On examination—speech thick, pupils normal, no optic neuritis, paresis of left cheek, tongue deviating slightly to left, complete palsy of left arm, anæsthesia of left hand. No less than seven cerebral tumours were found post-mortem.

(1) measuring 2 × 3 × 3 cm. in the right ascending frontal convolution at the level of the roots of the superior and middle frontal convolutions, and partly invading them, pressing also upon the ascending parietal. With this tumour the author connects the affection of the left arm and face.

(2) 15 cm. diameter, in the posterior part of the right temporo-sphenoidal convolutions near the angular gyrus. No affection of vision was complained of during life, but special examination of this point was not made.

(3) 15 cm. diameter in the middle frontal convolution, 4 cm. in front of (1).

(4) In the same convolution, but further forward, almost on the orbital surface.

All the above tumours involved the cortex.

(5) Size of a pea, in the white matter, just outside the right caudate nucleus.

(6) 2 cm. diameter, in the left ascending frontal convolution, but not involving the cortex.

[From the absence of right-sided motor symptoms the author concludes that this tumour had pushed the fibres aside without destroying them.]

(7) Size of a pea, colour deep black, in the right semilunar lobe of the cerebellum.

The tumours were of the nature of gliosarcomata; they presented considerable variety in their minute structure.

A case of Verbal Blindness with Right Lateral Hemianopsia. By T. A. MCBRIDE, M.D. (*American Journal of Neurology* as reflex or automatic, the muscular sense in the parts experimented upon supplying the stimulus to the centres whose action completes the required attitude.

Two Cases of Hysteria. By G. L. WALTON, M.D. (*Archives of Medicine*, August 1883).—1. Hysterical hemianæsthesia in a man, following injury. An engine-driver, æt. 55, who had never suffered from nervous symptoms previously, was thrown from his engine, rendered unconscious, and bruised on the right shoulder and hips; an emotional condition followed. When examined five months later, the right arm and leg were stiff and painful upon forced movements; there were adhesions round the right shoulder-joint: the general strength of the right limbs was impaired, without definite paralysis. Sensation was lessened over the whole body, principally on the right side; there was analgesia and ischæmia in the right arm.

Sight.—Concentric contraction of both fields of vision, but principally of the right; colour-vision normal in left eye, difficulty in distinguishing blue and green with right. V with left eye normal, with right $\frac{1}{10}$; fundus on both sides normal.

Hearing.—For watch, left ear $\frac{30}{60}$; with right ear, on contact only: tuning-fork through bone heard with left ear only, although the right meatus was then plugged with wax. Both tympanic membranes were thickened and retracted. The air-douche could not be felt in the right ear, although the Eustachian tube was pervious.

Taste and smell were wanting on the right side. The application of an electro-magnet relieved the anæsthesia, and improved the hearing on the right side: as the case improved, the hearing for low tones improved on the right side before the hearing for high tones. Apart from the accurate observation of the aural symptoms, the case is interesting as exemplifying the possible causation of hysterical symptoms in a healthy man by a railway accident.

2. Hysterical anæsthesia of special sense, accompanying a cutaneous hyperæsthesia.

An unmarried Portuguese girl, æt. 16, had suffered from pain and tenderness in the left breast for four months; the tenderness spread over the back and head and left arm. She was emotional, and had had one attack of unconsciousness. When examined, there was hyperæsthesia of the left side of the face, and head, chest, and of the left arm nearly to the wrist.

Vision.—Right eye $\frac{6}{5}$, left $\frac{1}{5}$, concentric retraction of the field of vision of the left eye, and with this eye blue was the only colour that could be distinguished. Nothing abnormal in either fundus.

Hearing.—Right ear normal; left ear nil, whether for bone or air-conduction; the membranes were normal on both sides. Smell and taste absent on the right side.

The author gives this case as an exception to the rule that anæsthesia of a special sense accompanies that of the integument covering the organ of special sense. Previous investigations by him ('BRAIN,' No. 20, 1883) have established this fact with reference to the ear. In the present case there was hyperæsthesia instead of anæsthesia.

Hysterical Locomotor Ataxia. By M. PUTNAM JACOBI, M.D. (*Archives of Medicine*, February 1883).—A widow, aged 35; father and brother insane; history of uterine disturbance. Vague nervous symptoms following a fright: three years after this, difficulty in going up and down stairs; finally unsteadiness in walking; but no pain. Slight improvement when the uterine disease was treated. After six months a staggering gait, the foot being thrust incoherently forwards and then brought down suddenly. Movements of legs not ataxic, when in the recumbent position. Absence of patellar tendon-reflex. Slight unilateral optic atrophy, but this apparently was due to injury. Attacks of dysphagia and dyspnœa; still later, shooting-pains from the hip to the ankle. Improvement followed an operation on the cervix uteri: but cramps, twitchings, and paresis of the limbs came on, and got so bad that she could not go up and down stairs at all. During the next two years she was away travelling, and improved very much. Four and a half years from the date of the first observation all her symptoms had disappeared, there was only a very slight swaying in the gait. But the tendon-reflex was still absent.

On an authentic Case of the disappearance of the Tendon Reflex without ascertainable Pathological Basis. By E. C. SPITZKA, M.D. (*American Journal of Neurology and Psychiatry*, August 1883).—A medical man, aged 32, had been used in boyhood to produce the patellar tendon-reflex in himself for amusement. It disappeared, according to his account, between his eighteenth and twenty-fifth year. At this period he drank heavily. He paid no attention to the disappearance of the reflex, till he happened to see the author examining a patient in this respect. The author verified the absence of the reflex, and considers its appearance in an individual otherwise sound to be unique. It is said, however, that the gait was peculiar, that he was cautious in walking in the dark, and that there had been occasioned cramps of the left gastrocnemius, and startings of the feet. [The absence of spinal disease would therefore seem to need verification at a later date.]

Arthropathies in General Paralysis of the Insane. By Dr. J. C. SHAW (*Archives of Medicine*, April 1883).—The author limits his observations to spontaneous lesions of the joints or bones, occurring without fracture or injury.

Case 1.—General paralytic, with grand delusions, ataxia of movement, absence of tendon-reflex. Duration of disease estimated at three years. Lameness of the right leg was observed; the cause could not be satisfactorily made out during life. Subsequently a similar lameness appeared in the left leg. There seemed to be no pain. Fracture or dislocation of the femur, due to trophic bone-lesion, was conjectured to be the cause. *Post-mortem*, there was found marked absorption of the head of the femur, the entire upper part was more spongy than natural; there was a deposit of bone below the trochanter, having a separate development in the periosteum, and not connected with the femur. There had been a backward dislocation caused by the absorption of the head of the bone.

Case 2.—Dementia, hesitating speech, tremor of lips and tongue. No delirium of grandeur, tendon-reflex normal. Redness and swelling occurred first in the left middle finger; then in the ring-finger; finally in the small joints of all the fingers. There was crepitus on passive movement, and actual dislocation of some of the joints. No post-mortem.

Case 3.—Hesitating speech, extravagant ideas; slight fibrillary tremors. Small sores appeared on the legs. A swelling of the right lower jaw appeared, which on examination proved to be due to necrosis of the bone; the teeth had fallen out; there were two fistulous openings. There appeared to be solution of continuity of the bone, as crepitation could be felt. There was no pain, and the skin in the neighbourhood was anæsthetic. A month later he began to limp on the right leg. Redness and swelling of the hip was discovered and an abscess opened. It was surmised that trophic changes were going on in the joint.

Case 4.—Advanced dementia, stammering speech, unsteady gait, some rigidity of muscles, with exaggeration of tendon-reflex, epileptiform attacks. No symptom of osseous lesion was observed during life, but post-mortem the right hip-joint was found to be the seat of commencing trophic disturbances, the head of the femur was beginning to undergo absorption, and would evidently have terminated in the same condition as that in Case 1.

The author considers these cases to be instances of arthropathy, such as has been described by Charcot and others in locomotor ataxy. He thinks that trophic lesions of the bones will be found in many diseases of the nervous system, and that probably lesions in many regions of the cord and brain give rise to such trophic disorders.

Locomotor Ataxia, terminating in General Paralysis of the Insane. BY CHARLES K. MILLS, M.D. (*Journal of Nervous and Mental Diseases*, July 1883).—A man, æt. 47, had for three years suffered from so-called rheumatism; darting pains, tingling, numbness, &c., in feet and legs, and afterwards his hands. He had had a chancre, without secondary symptoms, many years previously. On examination, no actual paralysis, but inability to stand with heels together or eyes shut, girdle pain, awkwardness of hands. No mental symptoms. Much improvement during seven months treatment; then, after a few months, permanent relapse, pains severe, gait staggering, anæsthesia of hands and feet. In two years more (five years after his first symptoms) his ideas became "queer and lofty"; in a year more, well-marked delirium of grandeur, peculiarities of speech, tremor of tongue and lips. In another year he had to be sent to an asylum, where he died demented, eight years after the first appearance of the "rheumatic" pains. The intra-cranial pia mater was opaque and congested over the motor area of the brain; the frontal and occipital convolutions were flattened. The spinal pia mater was thickened and the cord irregularly shrunken. Summarising the results of his microscopic examinations, the author says that the posterior columns (both columns of Goll and posterior root-zones) showed well-marked sclerosis in their whole length, best marked in the lumbar region, and diminishing from below upwards; lepto-meningitis was present everywhere. The medulla oblongata was much sclerosed on one side, and slightly so on the other. Sclerosis was also found in the pons, crura, optic thalami, and such parts of the cerebellum and convolutions as were examined.

The Early Symptoms of General Paralysis of the Insane. By Dr. W. B. GOLDSMITH (*Archives of Medicine*, August 1883).

—An analysis of 100 cases of general paralysis, in which the initial symptoms were reported to the author on competent authority, gave the following results:—

Eighty-seven cases were men, thirteen women. Of *physical symptoms*, the earliest were defect of articulation in 38 cases (usually a hesitancy of speech, recognised best during quiet conversation, with occasional elision of a syllable); some form of seizure in 20 (epileptiform in 13, like petit-mal in 4, apoplectiform in 3); tremor of lips and face in 8; inco-ordination of gait in 10; diminished sexual power in 6; general tremor in 6; cutaneous numbness and tingling in 3; changed cheirography in 2; vasomotor changes, hyperidrosis, localised cutaneous hyperæsthesia, general cutaneous hyperæsthesia, each in one case; ptosis, with external squint, diplopia alone, failure of sight from optic-nerve atrophy—each once (in syphilitic cases). Nine patients had also suffered from pain in the head, previously to the symptoms here enumerated.

The relative frequency of certain other symptoms (such as would escape a non-medical observer) is given, as they existed when the patients were admitted into the hospital (this was at very variable periods of the disease); thus the *patellar tendon-reflex* was normal in 46 cases, supra-normal in 24, very marked in 14, very slight in 12, absent in 4; the absence of change therefore in this phenomenon does not render the disease improbable; well-marked exaggeration in both legs is corroborative evidence of the disease; diminution or absence is evidence, but less valuable evidence, of the disease. Particular conditions of the patellar tendon-reflex cannot be connected with particular stages of the disease. Disorder of gait always accompanied absence of the tendon-reflex. The *pupils* were unequal in 16 cases, both small in 6, both dilated in 4. Inequality of pupils is therefore not so common as is generally stated. There is no particular state of pupils characteristic of a particular stage of the disease.

Of *mental symptoms*, the earliest were: failure of capacity (usually loss of memory) in 36 cases; depression in 22; exhilaration and self-satisfaction in 17 (accompanied by erotism in 9); insane delusions in 25 (delusions of wealth and grandeur in 12 cases). In the women the mental symptoms were usually less pronounced and less active than in the men.

As regards the relative time of appearance of the two classes of symptoms (mental and motor), in 68 cases they were noticed simultaneously; in 24 the mental first; and in 8 the motor. Without guaranteeing absolute accuracy in these observations, the author thinks the two classes of symptoms generally appear about the same time.

His general conclusions in addition to those already given are as follows:—that it is exceptional to find the striking and characteristic group of symptoms described in most text-books; that the earliest and most characteristic motor symptom is defect of articulation; that changes in the pupils and disorders of gait are less frequent, and of less diagnostic importance than is usually supposed; that hallucination or impaired function of the special senses is very rare as an early symptom; but that attacks of convulsions or coma constitute an early symptom of frequency and importance; that among mental symptoms the earliest is usually depression, with simple failure of mental capacity and activity.

Cases of Locomotor Ataxia. By S. G. Webber, M.D. (*Journal of Nervous and Mental Disease*, July 1883).—These are intended each to illustrate some unusual feature of the disease. The clinical histories are not very perfect.

Case 1.—During 20 years severe attacks of flatulence and pain, chiefly in the right side, without any known cause; sometimes with nausea, rarely with vomiting. Except absence of patellar tendon-reflex, there was no other symptom.

Case 2.—"Contractions" in the chest, causing difficulty of respiration.

Case 3.—Neuralgia of the occipital and trifacial nerves, with glycosuria.

Case 4.—Deafness, for which the condition of the ears was insufficient to account, beginning with an attack of vertigo.

Cases 5 & 6.—Preservation of patellar tendon-reflex.

Case 7.—Return of patellar tendon-reflex, which had at first been absent, together with improvement in other symptoms.

Case 8.—Well-marked symptoms of ataxy (including absence of patellar tendon-reflex) reappearing and disappearing several times within a year.

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END OF VOL. VII.

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